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NEUROLOGICAL DIAGNOSTIC CRITERIA IN DISEASES OF THE BRAIN

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A discussion of diagnostic points in diseases of the brain is always apropos when one considers the frequency with which these conditions, often with diffuse, varied, and puzzling symptomatology, are met, their usual seriousness and grave prognoses, and consequent difficult management and treatment.

Much clinical and laboratory work is constantly being reported which adds to our fund of knowledge regarding brain diseases and offers aid in their more accurate diagnosis and subsequent more able treatment. Studies in brain pathology have assisted in clearer understanding of the signs and symptoms which arise in cerebral vascular changes and accidents and in the various localized and diffuse infections and degenerative processes of the brain and its coverings. Examinations of the cerebrospinal fluid with pressure readings, frequently offer differential aid while encephalography and ventriculography are often most helpful in differentiation and localization of brain pathology.

Animal experimentation by Ranson, Davis, Pollock and others, has added to the knowledge of conditions arising from basal ganglion, cerebellar and midbrain disturbances.

The neurosurgeon and neuropathologist have contributed much the past few years with their close correlation of operative disclosures with pathological studies, which give more accurate and helpful information by means of the newer nerve-cell-staining methods of Hortega and vital stains of frozen sections.

The attempt has been made in this presentation to offer practical aids in differential diagnosis, without specialized data which would more aptly apply in the highly specialized clinics having the assistance of completely equipped laboratories in charge of highly skilled workers, and the frequent consultations of highly trained confreres in the associated specialties of medicine and surgery.

CIRCULATORY DISTURBANCES OF THE BRAIN

1. Cerebral Anemia
   - Acute and chronic
   - Local (intermittent claudication)
2. Cerebral Hyperemia
   - Temporary and prolonged
   - Sunstroke
3. Acute Cerebral Vascular Lesions
   - Thrombosis—Hemorrhage—Embolus
   - Pseudobulbar Palsy
4. Acute Bulbar Palsy
   - (Posterior inferior cerebellar artery syndrome)
5. Cerebral Arteriosclerosis

Cerebral symptoms following extensive loss of blood are difficult to differentiate from shock, but increasing evidence favoring vasomotor vessel innervation allows the possibility of both general and cerebral anemia.

In mild acute anemia the patient has air hunger and syncopal attacks, and complains of noises in the ears and spots before the eyes. The skin is pale, cold, and moist; respiration is rapid, shallow, and irregular; and the pulse is rapid and of poor volume; usually being secondary to cardiac impairment or sudden mental shock. When more severe, as from extensive sudden loss of blood, the onset is sudden, with roaring in the ears, marked weakness, dizziness, possibly nausea and vomiting, apathy followed by deepening coma and death, if the blood loss is sufficiently great. The same progression of symptoms may occur in heart failure with sudden loss of position.

In chronic anemia, as seen in leukemia, pernicious

anemia, cachexia, or repeated loss of blood, similar varying symptoms may occur, with drowsiness, apathy, and the expression of delusions or hallucinations. General supportive measures and treatment of the underlying causes are indicated.

Local cerebral anemia due to transient spasm and closure of an artery temporarily deprives a part of the brain of its blood supply, with symptoms demonstrating the functions of the area involved. This condition may result from intermittent claudication of the cerebral vessels, as is sometimes seen in cerebral arteriosclerosis, from pressure of a tumor, may occur in endarteritic or sclerotic diseases of the cerebral vessels, or present itself before the onset of an actual cerebral thrombosis. Transient symptoms, as aphasia, momentary facial twitchings, temporary palsies and sensory disturbances, may thus appear long before more severe lesions cause permanent evidence of cerebral impairment.

Temporary cerebral hyperemia may result from severe exercise in hot weather, alcoholic intoxication, inhalation of some gases, intense excitement, or sun stroke. Chronic hypertension and polycythemia may cause a more prolonged congestion of the cerebral vessels, when the usual symptoms of flushed face, throbbing temples and ear drums, a sense of head fullness and warmth, dizziness and headache, with occasional disturbances of consciousness, insomnia, temporary palsies, and, rarely, convulsions, may occur.

Acute Vascular Lesions of the Brain (Apoplexy). When an acute cerebral vascular insult occurs, the first important factors to determine are the type, extent, and location of the lesion, in the order named. Determination whether the condition is due to thrombosis, hemorrhage, or embolism, decides the immediate treatment to be instituted. Contrary to usual opinions, cerebral thrombi occur more frequently than hemorrhages. A thrombus is more liable to occur when the general circulation is at its lowest tension, while hemorrhage is more likely following exertion and physical or emotional strain. Jorgenson[11] after lumbar punctures in 140 patients with cerebral hemorrhage or thrombosis, concludes cerebrospinal fluid pressure above 300 mm. of water almost always indicates hemorrhage; also hemorrhage or xanthochromic fluid and increased amount of protein, even when the fluid is not bloody or yellow, indicate hemorrhage. As cerebral hemorrhage is more frequently soon fatal in spite of attempted remedial measures, without definite evidence to the contrary, the more favorable results are likely from treatment of the condition as a thrombotic process, with recumbent position, external heat and intravenous glucose. If the blood pressure is high and the left heart embarrassed, venesection may be indicated, and so-called cardiac stimulants withheld.

On the other hand, stimulation of the circulation, sedatives reducing restlessness, and the lowered head, may lessen the amount of post-thrombotic suffering, and limit further ensuing cerebral damage which might occur. Also hemorrhage may follow thrombotic softening, the danger of which is increased by restlessness and hyperactivity. With increasing hemorrhage, repeated lumbar punctures and glucose intravenously lessen the intensity of pressure upon the brain[12] with its resultant cerebral damage, irritation causing severe head pain, delirium, mental confusion and clouding and the onset of coma, and death, if it is sufficiently long continued. Kennedy and Wortis[13] state that caffeine sodium benzoate, hypodermically or intravenously, reduces increased intracranial pressure promptly.

It is doubtful if cerebral embolism may occur without endocarditis—from vegetations on the heart valves, particularly in mitral stenosis, or, more rarely, from a small embolus dislodged from the pulmonary vein or the aorta. Part of a thrombus in a larger vessel may break off and lodge in a more terminal cerebral vessel, and still more rarely a calcareous particle may be dislodged from the atheromatous walls of an artery. Occasionally bacterial emboli, circulating in the blood stream, may lodge in a cerebral vessel, but encephalitis or brain abscess is then more likely to result.

It is questionable whether any prophylactic treatment of cerebral vascular lesions is effective, except antisyphilitic treatment where that disease is present, though syphilitic thrombosis occurs despite vigorous antiluetic treatment. Repeated lumbar puncture is indicated when blood appears in the spinal fluid, particularly in intraventricular bleeding, in which condition signs and symptoms are usually rapidly and seriously increasing unless the pressure is thus lessened.

The extent of cerebral involvement is usually shown by the severity of symptoms—coma lasting over 24 hours, deepening coma, hyperpyrexia or markedly subnormal temperature, indicating severe cerebral injury and a poor prognosis. Cerebral hemorrhage[10] probably does not cause immediate death—that is, in less than one hour—even when the hemorrhage is into the pons or ventricles.

As to location, no part of the brain is exempt from

thrombus or hemorrhage, and few from embolus. The most frequent locations for hemorrhage are in the lenticulostriate artery—the artery of apoplexy, and in the lenticulo-optic artery. Emboli usually lodge in terminal vessels or in the angles of bifurcation of the carotid, vertebral and sylvian arteries, serving as points for thrombotic processes.

The middle cerebral artery is frequently occluded. Hence, the most common brain involvement is of the external and internal capsules, the thalamus and striate body. The usual ensuing hemiplegia is a corticospinal tract lesion which frequently may be differentiated from the more transient paresis of diabetic and uremic coma, acute toxic condition, alcohol, and so forth, cerebro-arteriosclerosis, encephalitis, syphilis, and paresis, with the aid of appropriate laboratory tests and available history. In determining the location of the lesion from the paralysis shown, and in differentiating it from other types of paralysis, it is helpful to remember: (1) movements, not muscles, are affected; (2) naturally, unilateral movements are most affected; and (3) more highly specialized movements are more affected than those more automatic; for example, impairment of emotional facial movements is not noted, but voluntary facial movements show the weakness; eye closure may be proper, but resisted lid movements are poorly accomplished; also, the patient may be unable to close the eye on the affected side without the other closing at the same time. The protruded tongue may deviate toward the side of the lesion, but phonation, mastication, and deglutition are properly performed. Neck and trunk movements are little affected.

Involuntary movements occasionally occur in vascular hemiplegia, as does also pain, which is usually arthritic, due to the fact that greater strength of the flexors than of the extensors results in contractures; bu the pain may be thalamic in origin. In left-sided cerebral lesions in right-handed individuals, some form of aphasia is usually present with the right hemiplegia, due to the proximity of Broca's area, the center for speech, to the motor area. Crossed hemiplegia may occur with a lesion in the quadrigeminate body, shown by a third-nerve palsy with paralysis of the face, arm, and leg of the opposite side. In the pons with a branch of the basilar artery involved, there may be paralysis of the face on one side and of the arm and leg on the opposite. Double hemiplegia may result from a single lesion in the pons where the two pyramidal tracts are in close proximity, or from two lesions in the internal capsules of both hemispheres, the latter giving the syndrome of pseudobulbar palsy—bilateral hemiplegia with dysarthria, dysphagia, impaired mastication, involvement of the truncal musculature and impaired sphincter control.

Acute bulbar palsy is usually due to thrombosis or hemorrhage of the posterior inferior cerebellar artery, manifested, if not sufficiently severe to be rapidly fatal from cardiac or respiratory failure, by flaccid lower-motor neuron paralysis of the lower cranial nerves, with dysarthria, dysphagia, paralysis of the palate, pharynx, larynx, and tongue. With the lesion more cephalad, contralateral monoplegia, bilateral hemiplegia, homolateral cerebellar ataxia, varying sensory symptoms as bilateral facial anesthesia, on account of each sensory trigeminal descending root containing fibers for both sides of the face, may occur, depending upon the location and extent of the involvement. Obviously, great care is necessary in feeding a patient with paresis of the throat muscles, to avoid an aspiration pneumonia.

Cerebral arteriosclerosis gives primarily psychotic symptoms, but should be included in a discussion of cerebral circulatory disturbances particularly to emphasize two points: (1) the fact that it may exist without evidence of general, or elsewhere localized, arteriosclerosis, and may be localized in but one, or a portion of one, cerebral vessel, and (2) that it should be considered in individuals past forty who complain of headache, dizziness, noises in the ears, lack of ability to concentrate, memory impairment, irritability, emotional instability, restlessness, and similar symptoms, with or without hypertension, where there might be a tendency to diagnose the condition as functional (psychoneurotic). The progression of symptoms may be manifested by transient palsies, aphasia, abnormal movements and sensory symptoms. Hallucinations and delusions, particularly of infidelity, may occur; memory may become much impaired, followed by defects of all the higher faculties, and progressive mental deterioration to dementia resembling senile dementia in many respects, may follow. But usually it appears at an earlier age, and is more acute in onset and rapid in progression, with a tendency to focal signs of involvement. The fundal vessels may be quite sclerotic. Pathologically numerous disseminated areas of softening and atrophy are found, with necrosis, fatty infiltration, senile plaques in the cortex and degeneration of nerve cells.

Alzheimer's disease, or presenile psychosis, is a severe, rapidly progressive dementia, occurring at an earlier age than senile dementia, characterized by marked memory defect, disorientation, hyperactivity,

and, frequently, irritability, probably arteriosclerotic in origin.

MENINGITIS

1. Epidemic cerebrospinal.
2. **Tuberculous.**
3. Purulent.
4. Acute serous.
5. Acute spinal leptomeningitis.
6. Chronic cerebral leptomeningitis.
7. Acute spinal meningitis.

The brain and spinal cord are completely covered by the leptomeninges, the pia and arachnoid, closely approximated, continuous structures separated by the space containing the circulating cerebrospinal fluid, which is continuous with the ventricular system, surrounds the convexity and base of the brain and extends around the spinal cord. Consequently, an acute bacterial infection starting anywhere along this communicating system, may spread rapidly throughout its extent, but usually more extensively affects a given area, especially the base of the brain in the interpeduncular space, over the cerebral hemispheres or the free surfaces of the cerebellum and pons. The process usually becomes widespread, however, and allows an early diagnosis by lumbar puncture, which sometimes is the only means of determining the presence, but particularly the type, of the meningitis.

The prominent symptoms noted at the onset, in a number of cases of epidemic cerebrospinal type reviewed, were: headache in 50 per cent, often quite severe and continuing as long as ten days before onslaught of the disease; vomiting in 43 per cent—frequently of several days' duration; pains in the back of the neck, back, or extremities, or general soreness and aching, in 30 per cent; sore throat in an equal number; listlessness and general malaise and apathy in 20 per cent; chills in 14 per cent; and fever in 22 per cent. The illness began with delirium in 3 per cent, stupor in 6 per cent, and convulsions in one epileptic.

The neurological signs of pathology noted in these cases were: stiff neck in 87 per cent, bilateral Kernig in 50 per cent, and unilateral in 7 per cent, bilateral Babinski in 21 per cent, unilateral in 3 per cent, Brudzinski in 27 per cent, contralateral reflex in 7 per cent, opisthotonos in 21 per cent, retraction of the head in 10 per cent, and absence of pupillary reaction in 33 per cent. Ptosis of one eyelid was presented in 13 per cent; strabismus, nystagmus and corneal anesthesia were each noted in 7 per cent. The deep reflexes were generally increased early in the disease and later decreased. General rigidity was noted in 7 per cent, while 3 per cent showed rigidity of the upper extremities, and 3 per cent on one side. Spasticity was evidenced in 17 per cent; 14 per cent evidenced pain on motion of the neck, the same number general twitching, and 7 per cent tremors of the tongue and hands. Photophobia was distressing in 7 per cent, urinary incontinence in an equal number, and general hyperesthesia was marked in 3 per cent. No neurological signs of pathology were elicited in 7 per cent—very fulminant cases.

Many of the signs enumerated occur in every case of meningitis, the differential diagnosis as to type often needing to be determined by the evidence found on examination of the spinal fluid. Clinically, tuberculous meningitis is always secondary to tuberculosis elsewhere and is more insidious in onset, usually showing prodromata for at least a number of days, and possibly several weeks, though the onset may be acute, with convulsion, agitated delirium and progressive coma. The disease usually lasts several weeks, with an almost invariably hopeless prognosis, those recovering from the condition diagnosed clinically as tuberculous meningitis likely being toxic meningitis, meningismus or mild meningeal signs sometimes seen in any infectious disease, only differentiated by the spinal puncture, and the presence or absence of the tubercle bacillus.

Purulent meningitis is meningeal infection with pyogenic micro-organisms, most commonly from otitis mastoiditis. Skull fracture may be followed immediately by acute meningitis and, rarely a traumatic scar may harbor infectious organisms which may result in a purulent meningitis after a long interval of health. Infections from the nose, sinuses, and throat, carbuncles of the head and neck, influenza, and whooping cough, and, more rarely, typhoid fever or pneumonia, may be complicated by purulent meningitis, the last being particularly virulent in this usually fatal disease.

Acute serous meningitis may occur as a complication of purulent otitis, or during the course of infectious diseases, closely simulating purulent meningitis. Rapid recession of symptoms usually follows spinal puncture, with recovery hastened by repetition of the procedure; but acute internal serous meningitis with obstruction and dilatation of the ventricles, may become chronic or remitting and be followed by internal hydrocephalus.

Acute spinal leptomeningitis is rarely a separate clinical entity, the inflammatory process usually quickly spreading generally over the extensive areas of the meninges. When it does occur, the course is generally fulminating, with death resulting in a few

days; but, if the process remains localized in the cord, recovery may occur, with usual residual paresis and radiating pains from spinal root involvement.

Chronic leptomeningitis, excluding syphilitic meninges infections, may rarely occur as an atypical protracted form of meningococcic or purulent meningitis, or in chronic alcoholism and lead poisoning, chronic cysticercus infection and senile dementia; but it is doubtful whether it can be recognized as an independent clinical entity without an exploration.

In acute meningitis, where diagnosis may be difficult, the cerebrospinal fluid examination may be differentially diagnostic. The cell count in tuberculous meningitis shows 20 to 100 small lymphocytes, while in diffuse purulent meningitis hundreds to many thousands of polymorphonuclear leukocytes may be present. In epidemic encephalitis the cell count is low—from normal to 30 lymphocytes, in cerebrospinal lues, 50 to 500, and in paresis 30 to several thousand. The protein content of the cerebrospinal fluid is four plus in diffuse meningitis and in the tuberculous type, absent or present in slight amount in epidemic encephalitis, 4 plus in paresis, and 3 plus in cerebrospinal syphilis.

In tuberculous meningitis the chloride content may drop to 400 to 600 mgm. per 100 cc. when it is diagnostic. In epidemic encephalitis there is an increase above the normal of 750 mgm. per 100 cc., and in diffuse meningitis a slight reduction of chlorides and absence of sugar.

Tuberculous meningitis shows a normal or slightly decreased, and epidemic encephalitis normal or slightly increased, sugar content above the normal average of 45 mgm. per 100 cc.

Cultures from the cerebrospinal fluid determine the type of meningitis, whether meningococcic, streptococcic, pneumonococcic or influenzal, staphylococcic meningitis being extremely rare. In tuberculous meningitis the tubercle bacillus may be found in the sputum or by guinea pig inoculation. Cultures from the spinal fluid of epidemic encephalitic patients are sterile.

The treatment of acute meningitis is unsatisfactory and often futile, being essentially symptomatic except in epidemic cerebrospinal type, where antimeningococcus serum should be administered early in large doses intraspinally, intravenously and also intramuscularly. In fulminant cases, where death may oc-

Cerebro-Spinal Fluid in Differential Diagnoses

DISEASE	CELL COUNT	PROTEIN CONTENT	CHLORIDE CONTENT	SUGAR CONTENT	WASSERMANN REACTION (Sp. Fl.)	COLLOIDAL GOLD REACTION
Normal	3 - 8 (to 10)	None (.018%)	750 mgm. per 100 cc.	45 mgm. per 100 cc.	Negative	
Diffuse Purulent Meningitis	Hundreds to many thousands polymorphoneuclear leukocytes	+ + + +	Slight reduction	Absent		
Tuberculous Meningitis	20 - 100 small lymphocytes	+ + + +	Decrease to 400 to 600 mgm. per 100 cc.	Normal or slight decrease		
Epidemic Encephalitis	Normal to 30 lymphocytes	Absent or slight amount	Slight increase	Normal or slight increase		
Cerebro-Spinal Syphilis	50 to 500	+ + +			95-100% positive 80-90% in blood	90% Zone I
Paresis	30 to several hundred rarely	+ + + +			100% positive (Blood same)	90% Zone I
Tabes	20 - 200	+ +			90% positive (Blood in 85%)	90% Zone II

cur before evidence of signs, and the meninges show little involvement in the short time of the infection, death appears to be due mainly to the septicemia, and the clinical impression is one of an overwhelming blood infection. A number of deaths from epidemic cerebrospinal meningitis have been reported without meningitis[14] evidently due to meningococcemia.

In the cases reviewed, above mentioned, where the type of infection was particularly fulminating, as much as 115 cc. of antimeningococcic serum was administered intraspinously, 818 intracisternally and 100 cc. intramuscularly to one patient, without recovery. One patient, ill 23 days, was treated by fourteen alternating cisternal and spinal punctures, on account of apparent poor results in serum-treated patients, with some improvement, followed by recrudescence. Patients have been reported,[18] where little improvement followed intraspinal and intravenous administration of serum, who responded to administration of 30 cc. daily for a week into the lateral ventricle following trephining. Goldman and Bower[20] compared the results of treatment in fifty patients treated by the cisternal route and forty-eight by the lumbar, with a mortality rate of 25.5 per cent by the former method and 52.3 per cent by the latter. Evans and Welsh[17] used 5 cc. of antimeningococcic serum in each common carotid artery and 10 cc. in the cisterna magna with favorable results. They prefer the cisternal route to the lumbar, avoiding spinal block, and, adding some air with the injection, they believe the amount of arachnoiditis is much lessened. The intracarotid administration of Pregl's iodine and acriflavine has been used by Crawford,[21] Kolmer,[22] Dowman, et al, with some success. Reports indicate favorable results are directly proportional to the amount of serum used and the earliness of its use. More frequent spinal drainage is widely urged, some experienced writers advising drainage every six to eight hours until marked amelioration of the symptoms. Where the serum being used does not seem therapeutically effective, change to another manufacture is advised—titration and other laboratory tests of serum efficiency being reported as nondecisive of the clinical value of a particular serum. Large amounts of serum used earlier, more extensively and frequently, with more frequent brain dehydration and drainage, should show better results than present mortality statistics indicate. The incentive for more desirable results is ever present when one sees the residuals in patients recovering with deafness, blindness, mental abnormalities, and paralysis, and it is realized that the mortality rate gives little conception of the seriousness of this infection.

In the post-meningitic symptoms, routine repeated lumbar punctures are often effective, particularly in relieving the severe handicapping recurrent headaches that frequently remain as a residual for many years. In one patient who came to the writer's attention, 17 years after an extremely severe infection, with temporary blindness, deafness and hemiplegia, and ocular palsies, all were recovered from except the extremely severe, persistent headache every two to five weeks.

Recovery from pneumococcic meningitis, which is considered almost invariably fatal, is reported by Max Weinberg[18] following the use of potassium permanganate enemata, using four ounces of a solution of a pint and a half of water containing two grains (1:5760) of potassium permanganate.

Gonococcus meningitis is reported[9] in a patient following five weeks' illness, with no response to antimeningitic serum, rapidly improving to recovery after three weeks' administration of antogenous vaccine. Another case is reported,[9] however, which improved rapidly with the administration of antimeningococcic serum given as non-specific therapy. Meningitis due to bacterium tularense[9] is reported, and the importance of prevention is stressed, as no therapeutic measures are known.

ENCEPHALITIS

1. In the course of a brain disease, e. g., meningitis.
2. As a variant, or in the course of acute anterior poliomyelitis.
3. With acute infections—measles, scarlet, pneumonia, etc.—and post-vaccinal.
4. Acute alcoholic polioencephalitis.
5. Polioencephalitis chronica inferior (Wernicke).
6. Acute serous encephalitis.
7. Epidemic encephalitis.
8. Encephalitis disseminata.

Encephalitis is an acute non-purulent inflammation of the gray matter of the brain, shown by some mental symptoms, usually meningitic signs, often pupillary and ocular disturbances, frequently some disturbance of motility, but most characteristically marked by signs and symptoms of multiple foci and pathology in widely separated parts of the brain. Hence, only a tentative classification may be attempted, as suggested by Wechsler.[3] (1) Encephalitis occurring in the course of any disease of the brain, as in the meningitides—syphilitic, tuberculous, men-

ingococcic and purulent. Purulent encephalitis of any extent merges into brain abscess. (2) Polio-myelo-encephalitis in the course of, or a variant of acute anterior poliomyelitis. (3) Encephalitis in the course of infectious disease—measles, scarlet fever, whooping cough, diphtheria, pneumonia, malaria, influenza, and in intoxications, and post-vaccinal, also known as acute hemorrhagic encephalitis of Strumpell, and more frequently occurring in children. (4) Polio-encephalitis acuta hemorrhagica superior of Wernicke, or acute alcoholic polio-encephalitis. (5) Polioencephalitis chronica inferior (Wernicke)—chronic bulbar paralysis associated with progressive muscular atrophy. (6) Acute serous encephalitis—usually a severe, extremely acute, toxic state, with transudation of serum into the perivascular spaces instead of cells. (7) Epidemic encephalitis, to which may be added (8) Spiller's encephalitis disseminata[19] characterized by progressive cranial nerve palsies, mono and hemipareses, sometimes bilateral.

Epidemic encephalitis, first described by Economo in 1917, following the great world-wide influenzal epidemic, has become pandemic, though somewhat milder (20 per cent mortality at first) possibly due to some mutation or alteration of the causative agent, or a developing immunity against the infection. Its most characteristic feature is, that of all acute diseases of the nervous system, it is the most widespread and disseminated, evidencing a diverse, varied and often puzzling symptomatology; but where an acute nervous symptom infection occurs without apparent cause, accompanied by some febrile reaction, possibly some signs of meningeal irritation, disturbances of consciousness, some radicular pains, abnormal involuntary movements, and ocular or pupillary disturbances, one is justified in diagnosing epidemic encephalitis.

With the diversity of symptoms, disseminated pathology is to be expected, and has been found, not only in the basal ganglia, particularly the substantia nigra and nucleus ruber, but also scattered through the cerebrum, in the thalamus, the cerebellum, and even in the medulla and pons.

The course of this disease varies as much as the diversity of its acute symptoms. The most distressing and handicapping symptoms—more tragic and incapacitating than the illness itself—may appear after a two to eight years' latent period, when the most common sequel appears in the form of the typical Parkinsonian syndrome, with progressive spasticity and rigidity of one side, followed by similar signs on the opposite side, postural disturbances, impairment of associated movements (dyskinesia), frequently pupillary abnormalities, the typical facies, rythmic tremors, occasionally oculogyric crises, tachypnea, and impulsive actions, frequently irritability, forgetfulness, lack of concentration and power to work, and occasionally actual psychotic manifestaions[20] with delusions and hallucinations, but particularly emotional episodes. In children sequelae are most frequently manifested in behavior and conduct disorders, marked emotionalism, impulsiveness, inability to integrate or concentrate well, and by decided personality changes, with cruelty and abnormal sexual tendencies, rather than the motor disturbances shown in adults. These disorders in children may be found to have closely followed an undiagnosed mildly febrile illness, with slowly progressive insidious symptoms, or without any observed prodromata having occurred.

Unfortunately, no effective treatment is available, but hyoscine hydrobromide 1/100 to 1/150 gr., two or three times a day, and tr. gelsemium, or tr. stramonium[21], in ascending doses, from 10 to 60 minims three times a day, have favorable effect on the spasticity and tremors and increase the feeling of general well being, in the Parkinsonian syndrome. Typhoid vaccine intravenously, producing protein shock, sometimes has temporary beneficial results. Levaditi's vaccine and a vaccine prepared by Drs. Stewart and Evans,[3] from cultures of a particular strain of Pfeiffer bacillus isolated and found in all post-encephalitic patients examined, have been used with reports of amelioration of signs in a number of patients. M. Lavai found relief to be instantaneous from intravenous injection of atropine in epidemic encephalitic hiccup—in most cases after a single injection, and only three of thirty-two proving refractory. Sudden relief has been recorded from painful injection of ether into the skin.

BRAIN ABSCESS

Pathology may result from (1) trauma by direct introduction, (2) middle-ear disease most commonly, and causative in one-half to one-third of the cases, (3) purulent infections elsewhere—e. g., bronchitis, empyema, pulmonary gangrene, (4) blood stream infection, rarely.

Abscess has been reported without bony vault injury following trauma, and after as long as 25 years' latent period.

Symptoms of brain abscess are those of an expanding space-taking inflammatory lesion—hence, both general and focal, but frequently masked by the underlying cause, as in mastoiditis or by the complicat-

ing meningitis, and may appear after a latent period of weeks or months, followed by a similar lengthy period of minor vague symptoms of headache, malaise, anorexia, apathy, and irritability which may be interpreted as neurotic. Tumor usually runs a slower course, is more likely to cause choked disk, and has not the history of preceding trauma or infection. Sinus thrombosis is characterized by high remitting fever, chills, positive blood culture, and, at times, a positive Queckenstedt test, but may coexist with abscess.

Circumscribed serous meningitis may simulate abscess, but usually disappears following lumbar puncture. The more acute and general signs and rapid course in meningitis, with examination of the cerebrospinal fluid, are usually differential. Vertigo and nystagmus are the rule in labyrinthitis, deafness is present, and usually with lack of response in the caloric tests, these offer diagnostic criteria. Diagnosis is often difficult, but when reasonably certain, exploration is justified. Ventriculography is contraindicated because of danger of rupture of a pus pocket.

INJURIES OF THE BRAIN

Varying symptoms result from skull trauma depending upon the force, direction, location, and nature, of the injury, with its ensuing damage to the cranial contents. Simple concussion may result without fracture in a physiochemical molecular alteration of the brain structures. Numerous minute hemorrhages, or even foci of softening, may occur. With a compound fracture there is grave danger of infection, with subsequent pyogenic meningitis, encephalitis, arachnitis, serous meningitis, epidural or deep-seated abscess—symptoms of the latter probably not occurring for weeks or months. Roentgenogram of the skull should never be omitted, for medico-legal reasons if no other. Epidural hemorrhage frequently gives a characteristic syndrome of a varying brief period of unconsciousness, followed by absence of symptoms for several hours or a day or more, with ensuing progressive somnolence, stupor and coma, focal tics and convulsions and hemiplegia.

In subdural hemorrhage the interval between the date of injury and onset of symptoms may be several weeks, when the effusion is found to be serous or serosanguineous. Where the hemorrhage is subarachnoid, the cerebrospinal fluid will be found bloody, and lumbar puncture should be repeated in the attempt to reduce cerebral edema—the effect of cerebral damage from pressure of the blood clot—and lessen the usual restlessness, delirium, and danger of cardiac and respiratory failure from paralysis of their centers in the bulb. Hypodermic magnesium sulphate 30 per cent or, better 50 per cent glucose solution, intravenously preferably, or rectally, also assists in reducing the cerebrospinal fluid pressure.

Kennedy[21] warns against too definite opinions in post-traumatic cranial symptoms and stresses the need for "harmony between the degree of injury, the subsequent history of the patient's symptoms and his physical condition; dissonance in this trilogy must be viewed askant and awake a suspicion of a suggested neurosis." He advises encephalography to assist in more ably determining the degree of brain injury and to estimate the organic disability in a particular case. He gives as criteria of brain injury sufficient to produce organic change in the brain:

A. Absolute Criteria,
   1. Roentgen evidence—skull fracture
   2. Bloody spinal fluid
   3. Bleeding from the orifices, especially from the ears
   4. Focal cerebral palsies.

B. Presumptive criteria in the order of their importance:
   5. Convulsive states proved to be posttraumatic
   6. Ventricular distortion proved to be posttraumatic
   7. History of prolonged unconsciousness
   8. History of adequate trauma, with especial consideration of the occurrence of vomiting following the injury.

Wilder Penfield suggests the use of oxygen rather than air, with much less ensuing post-injection headache.

BRAIN TUMOR

Tumor will but be mentioned for serious consideration where there is slowly progressively severe headache, at times recurrent cephalalgia without other obvious cause, occasional vomiting without nausea often projectile in type, often mental dullness, personality changes and dizziness, followed usually by focal symptoms of tics, Jacksonian attacks, convulsions, auditory and equilibratory symptoms, tremors, progressive paresis, visual and ocular signs and symptoms, and incoordination and hypotonia, particularly in the presence of choked disk.

Meningioma may be extremely slow in growth, with progression of signs and symptoms over a number of years, due to accommodation of the cranial contents to a slow-growing space-taking lesion, but spongioblastoma multiforme may be extremely rapid

in growth, with rapid and sudden onset of symptoms, as illustrated in a report[25] and review, by the writer, of seventy cases of this type where the average duration of illness from the first symptom—which in many was sudden, startling and severe in onset—was but 49 days.

Roentgen-ray of the skull offers but little help in diagnosis or localization; but ventriculography,[27 28] introduction of air into the ventricles after withdrawal of fluid, may reveal by roentgenogram their distortion, size, and shape, or encephalography[26] withdrawal of spinal fluid and injection of air at the lumbar spine, may assist in localization of the lesion.

Early diagnosis and surgical treatment is of prime importance to avoid progressive brain destruction, with permanent paralysis, blindness, or other focal incapacity, especially when untreated brain tumor is inevitably fatal. Frazier states that there are about four thousand patients with brain tumor in the United States, of which four hundred come to operation annually, and that diagnosis and treatment are improving.

SYPHILIS OF THE BRAIN

The pathological characeristic of neurosyphilis is its widespread dissemination in every portion of the cerebrospinal system, the parenchyma of the brain and cord, the nerves, meninges, and blood vessels, possibly invading a certain element and giving rise to a special clinical syndrome, but nevertheless evidencing some varying involvement in all other elements. This diffuse invasion limits definite classification, but the main focus or region involved allows grouping of neurosyphilis into vascular, meningeal, meningovascular and parenchymatous types. Clinical differentiation is often difficult, but distinction is necessary, both for treatment and prognosis, for paresis responds poorly to treatment and has a very poor outlook, in contrast to the much more hopeful prognosis in diffuse cerebral syphilis.

With the usual disseminated pathology present in syphilitic infection of the nervous system, the signs and symptoms vary greatly, but differ in no way from those caused by other cerebral lesions. Early in the disease, the vague, varying, fleeting, indefinite symptoms may simulate a neurosis, but with definite mental symptoms, evidence of motor symptom involvement, disturbed reflexes, paresthesias, ocular palsies, sensory changes or root pains, the diagnosis is usually indicated before laboratory reports are obtained.

The base of the brain is usually most involved, with resultant cranial nerve palsies, and, as the interpeduncular space is the favorite site of the attack, the optic and ocular nerves, particularly the oculomotor, frequently first evidence cerebral syphilitic disease. Unequal, irregular pupils responding poorly to light, very rarely may be present in chronic alcoholism or encephalitis, but are almost invariably diagnostic of neurosyphilis. Paralytic symptoms occur in cerebral softening from thrombosis or hemorrhage where the vessels are principally involved, and usually at a much earlier age than most cerebral vascular accidents. Examination of the cerebrospinal fluid is usually very helpful in differentiating the type of syphilitic involvement and paresis, due to the frequent resemblance of meningovascular syphilis, should never be diagnosed without a positive Wassermann reaction in both the blood and spinal fluid. In cerebrospinal syphilis the blood Wassermann reaction is positive in 80 to 90 per cent, and in the spinal fluid, 95 to 100 per cent positive. The colloidal gold reaction is diagnostic in 90 per cent or more of spinal fluids from patients with paresis or cerebrospinal syphilis. Paresis usually gives a slightly lower spinal-fluid cell count than in the cerebrospinal type, being thirty to several hundred rarely in the former and 50 to 500 in the latter.

Tryparsamide, used with the soluble bismuth preparations preferably, mercury rubs or injections, and the iodides, give the best results in neurosyphilis, but an uninvolved optic system should be present and two competent fundal examinations should be made the first month of its use, when, no toxic symptoms having shown in the eye, it should be quite safe to use indefinitely insofar as danger to vision is concerned. It should be given intravenously in 2 or 3 gm. doses every five to seven days, in a course of 50 doses, and the course should be repeated if the indications remain.

Paresis has shown definite response to fever therapy induced electrically, or by malarial infection, with 8 (to 14) chills, during which time arsenicals, except tryparsamide, need to be omitted. Courses of hot baths have apparently produced remissions, and protein therapy in the form of intravenous injections of typhoid vaccine in increasing doses of .2 to 2 cc. diluted with saline solution, produce temporary amelioration of excitement, agitation, restlessness, insomnia, and mental confusion and clouding. Gummata not yielding to usual antiluetic treatment for a reasonable time, should be treated surgically, as other brain tumors, particularly if they appear dangerous to life, or are causing incapacity or focal epilepsy.

DISORDERS OF MOTILITY AND DISEASES OF THE BASAL GANGLIA

1. Paralysis agitans (Parkinson's disease)
2. Progressive lenticular degeneration (Wilson's disease)
3. Pseudosclerosis of Westphal
4. Dystonia musculorum deformans
5. Huntington's chorea
6. Sydenham's chorea
7. Various athetoses, myoclonias, tremors and tics.

Disorders of motility are attributable to disease of the basal ganglia, mainly due to inflammatory lesions followed by degenerative changes. The manifestations and sequelae of chronic epidemic encephalitis may simulate practically all basal ganglia syndromes. Syphilis, tubercles, and vascular lesions of the basal ganglia, may be causative of abnormal motility, while manganese poisoning may give a syndrome characterized by coarse tremor, masked facies, bulbar speech and compulsive laughter, and carbon monoxide poisoning may be followed by the Parkinsonian syndrome from the softening caused in the lenticular muscles.

Disease of the striatum, as described by Wechsler,[2] is characterized by (1) rigidity, generally increased muscle tonus, (2) tremors and similar involuntary movements, (3) hypokinesia, decreased voluntary, especially spontaneous movements, (4) impairment of associated movements, (5) absence of "true" paralysis from involvement of the pyramidal tracts, (6) absence of sensory disturbances. Other signs are anomalies of posture, the body being held in flexion, and the commonly associated hyperkinetic syndromes —the choreas, athetoses, and dystonias. Definite localization of the lesions causing the various abnormal movements, is extremely difficult, due to the striate complex associations[29] of various parts of the striate body, including the caudate and lenticular nuclei—the putamen and globus pallidus, with the body of Luys, the substantia nigra and the red nucleus—between each other and with other parts of the brain—the cerebrum, cerebellum, the thalamus, the pons.

The more common syndromes included in basal ganglia disease are paralysis agitans (Parkinson's disease), progressive lenticular degeneration (Wilson's disease), pseudosclerosis of Westphal, dystonia musculorum deformans, various athetoses, Huntington's chorea, Sydenham's chorea, and a number of complicated involuntary movements, myoclonias, tremors and tics.

The syndrome of paralysis agitans is generally rarely recognized, especially since the more common occurrence of the Parkinsonian syndrome developing in chronic epidemic encephalitis. Pathologically, the classical type involves the globus pallidus and ansa lenticularis, mainly, while the post encephalitic involvement is more widespread and disseminated, particularly predominating in the midbrain, mainly in the substantia nigra and nucleus ruber, but also showing lesions in the cerebrum and also in some brains in the thalamus, cerebellum, and even in the pons. Also, the lesion in the latter is a chronic inflammatory change, originally, possibly followed by degenerative processes.

Clinical differentiation of the two types, even in older patients in whom only paralysis agitans is usually found, is made by experienced neurologists, but it is probably of little more than academic value. With the absence of history, symptoms and signs are usually more diverse in the post encephalitic variety, with some not formerly associated with the disease. Pupillary anomalies, ocular palsies, and, particularly, impairment of convergence from involvement of the third nerve nucleus, emotional instability likely from lesions in the thalamus, radicular pains, with loss of automatic and associated movements, are more prevalent in chronic epidemic encephalitis. Paralysis of upward gaze, respiratory anomalies, impulsive actions —particularly, sudden, forced, involuntary running, even with injuries from striking objects—bizarre movements and actions and personality changes, at times with psychotic manifestations, are symptoms frequently seen in the postencephalitic patient, not observed in the classical syndrome.

Progressive lenticular degeneration (Wilson's disease) is a chronic, progressive, fatal disease, usually familial, but neither congenital nor hereditary, resulting from bilateral symmetrical degeneration of the putamen of the lenticular nucleus, mainly occurring between the ages of 10 and 20. In this disease the liver is shrunken, shows multilobular cirrhosis and, later, a typical hobnailed appearance. Clinically, tremor, rigidity, contractures, mental deterioration to dementia, dysphagia, dysarthria, hepatic cirrhosis, progressive emaciation and the absence of evidence of involvement of the pyramidal tract, the sensory system or cranial nerves, characterize this hopeless syndrome.

Pseudosclerosis of Westphal resembles Wilson's disease and begins at about the same age, but the pathological process is more diffuse and less definite. Tremor and rigidity are characteristic, emotional disturbances are usual, with mental changes less severe than in progressive lenticular degeneration, ataxia occurs and occasionally optic atrophy, but the presence of a pigmented greenish ring one to two milli-

meters wide at the outer edge of the cornea (Fleischer and Strumpell's sign) is considered to be typical and pathognomonic.

Dystonia musculorum deformans consists in a slow, spontaneous, involuntary movement, following a definite pattern without purposeful end, shown by slow, twisting, turning torsion and irregular writhing and wriggling of the limbs and body. The symptom begins between 8 and 15, is usually very slowly progressive, beginning with a slow twisting, turning or flinging of the leg, apparently purposeless arm movements or torticollis, which may be considered hysterical, till the movements become widespread, involving the muscles of the back, neck, feet, and all of the leg muscles.

Double athetosis is a congenital affection, characterized by widespread mass movements of practically all muscles, particularly of the hands, feet and face. Speech disturbances are marked, and mentality is impaired.

Chronic degenerative hereditary chorea (Huntington's chorea). This disease is a hereditary, familial chronic, progressive chorea with mental deterioration terminating in dementia, usually beginning past 40. Most of the brain evidences miliary encephalitic foci with secondary atrophy and cell degeneration most marked in the cerebral cortex and basal ganglia. The choreic movements may first appear in the face or upper extremities, then become gradually more violent and widespread, with much facial grimacing and bizarre dancing and jerky gait, gesticulations and marked dysarthria, smacking lip and tongue movements, breathing anomalies and the steadily progressive mental deterioration. Cases have been reported without the mental deterioration, and others without the family history, but family history may be difficult to elicit, or be unknown to the patient or informants.

Chorea (Sydenham's chorea, St. Vitus dance). Sydenham's chorea is probably always infectious in origin and definitely related to the infections in tonsillitis, arthritis and endocarditis, usually occurring in the young, from 5 to 15 years of age, but not rarely as late as 25. Usually the disease begins insidiously, with restless, fidgety hyperactivity followed by the involuntary, spontaneous, jerky, irregular, purposeless movements, usually beginning in one limb, either upper or lower. The speech becomes jerky and explosive, and may become quite dysarthritic. Psychic symptoms, varying from restlessness and irritability to violent mania with hallucinations, may occur, as in other toxic-infectious psychoses. Very mild types may occur without the choreiform movements, the diagnosis being made from the history or presence of a tonsillitis, possibly with signs of an arthritic or cardiac infection, restlessness, general nervous instability and emotionalism, and the decreased deep reflexes. On the other hand, the illness may be very acute and terminate fatally in two or three weeks. Chorea gravidarum is especially serious, with a mortality given as high as 25 per cent. Treatment should be similar to that of endocarditis and arthritis without the cerebrospinal symptoms. Small's cardio-arthritides serum and antigen are very helpful in many cases. Sedatives, particularly by hydrotherapeutic means, rest, and supportive measures, are indicated. The prognosis is usually good, without residuals, but recurrences are common and the condition may become chronically intermittent. Usually, the older the patient, the more severe and prolonged the illness.

THE CEREBELLAR SYNDROME

1. Congenital—Cerebellar Agenesis
   Childhood Cerebellar Palsies
2. Acquired—
   (a) Acute Cerebellar Ataxia
   Traumatic
   Inflammatory
   (b) Hemorrhagic
   (c) Toxic-degenerative
3. Tumors

Synergia, or coordination, according to Mills and Weisenburg,[26] is the fundamental cerebellar function and dyssynergia, or ataxia and failure of proper coordinated movement, the fundamental symptom of cerebellar disease.

Disease of the cerebellum may be (1) congenital, as seen in cerebellar agenesis or hypoplasia and childhood cerebellar palsies, or (2) acquired as an (a) acute cerebellar ataxia, which may be traumatic or inflammatory, as in abscess formation or an encephalitic infection, (b) hemorrhagic, and (c) toxic degenerative, chronic toxic or arteriosclerotic with atrophy. Cerebellar tumors may give confusing signs from neighborhood involvement and pressure of the expanding lesion. In unilateral lesions, signs are usually seen on the homolateral side of the body, due to recrossing of the rubrospinal tract after efferent cerebellar fibers crossing to the opposite nucleus ruber.

The typical cerebellar syndrome manifests ataxia, dyssynergia, and decomposition of all active movements. The patient is broad based, swaying in any direction, and requires support for maintenance of the erect position. Gait is staggering and reeling,

with arms outstretched to reach support to avoid falling. Ataxia of the shoulder and pelvic girdles is present, with defective integrative synergy of the trunk and extremities. Incoordination is shown in all movements, dysmetria is shown in faulty measurement of rate, amplitude and force of a movement, and the patient is unable to execute alternating and successive movements rapidly and smoothly (adiadokocinesis). The knee jerk is frequently pendular. Nystagmus is common, ocular movements are irregular, jerky and oscillatory, with increased activity of the eyeballs, and speech is jerky, explosive, scanning. Hypotonia is usual, but no true paralysis is present, and the reflexes are unaffected.

SUMMARY

Practical diagnostic points in diseases of the brain have been presented.

1. A classification of cerebral circulatory disturbances has been offered, with some differential diagnostic aids, particularly as to type and location of acute vascular lesions.

2. The various types of meningitis have been discussed and the prominent symptoms at onset and most common neurological signs in their order of frequency in a number of cases of the epidemic type, reported. A comparative differential chart of spinal-fluid findings in the various types has been included and the methods of administration and reported results of different types of treatment, reviewed. Frequent brain dehydration and drainage and the early use of large amounts of serum early in the epidemic type, has been emphasized.

3. A tentative classification of encephalitis has been given and attention has been called to the diversity of symptoms in the chronic epidemic type and to its usual syndrome in children.

4. A few differential points in diagnoses of brain abscess, tumor, and the various types of traumatic cerebral hemorrhage, have been mentioned, and Kennedy's criteria of brain trauma given.

5. Diagnostic aid is offered in syphilis of the brain, by examination reports of the cerebrospinal fluid, and more liberal use of tryparsamide and fever therapy has been advised.

6. Disorders of motility are due to basal ganglia disturbances and the usual resulting syndromes from their impairment and the cerebellar syndrome, have been briefly described.

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DISCUSSION

DR. A. B. STEWART (Las Vegas, N. M.): Dr. Irish, in addressing this Society, the members of which he has not heretofore met, has (if I may use a bridge term) left no point vulnerable for discussion. His paper covers such a vast field that it is very difficult even to attempt a discussion. There are only one or two points which might be stressed a little more, and one is spinal drainage. To the general man, spinal puncture is no longer a procedure that clarifies him in any particular, and it is the one thing which is going to aid the individual most in a great many diseases of the brain. Dr. Irish spoke about tryparsamide given intravenously in neurosyphilis, but it all depends upon the condition of the patient whether it should be used first or last or in conjunction with fever therapy. The doctor gave a beautiful resume of diagnostic criteria of all diseases of the brain and has left himself open to absolutely no criticism. If he had shown us a few cases, we might have been able to do so, but, as it is, his paper is so complete he has given us no chance. His classifications are very good. The paper should be read more than once in order fully to grasp the magnitude and thoroughness with which he has covered the field.

DR. A. H. VOGT (Albuquerque, N. M.): I am glad to have had the opportunity to listen to Dr. Irish's excellent and exhaustive paper. He did not leave himself open to attack and there is not much to say in discussion. The treatment of neurosyphilis falls very much into three groups. One third, more or less, will be benefited by the use of malaria therapy; the second group, by the use of tryparsamide of bismuth; and the third group, by the old-fashioned spinal puncture, which now seems to have gone out of use. We should consider seriously the economic loss caused by this disease, and that brings up the question of complete treatment of syphilis. In a recent issue of the Journal of American Cutaneous Review, there appeared an article by a foreigner, who asked if treatment prevents syphilis, or aggravates it. For the protection of society we must treat syphilis and urge treatment in an endeavor to prevent neuro-syphlis.

DR. F. C. VICKERS (Deming, N. M.): I should like to ask Dr. Irish's opinion of electric thermotherapy.

DR. IRISH (closing): It is generally conceded by many men whose opinions are much worth while, that electric thermotherapy has not yet become as acceptable as other forms of thermotherapy, or fever therapy. Hot baths give some temporary relief in quieting patients and relieving and limiting agitation, restlessness and insomnia. I am very appreciative of the discussion, and of the way my presentation was accepted. This subject has been rather a large bite to take, but I thought it might possibly interest some of you enough to stimulate neurological reading. Most men do not read neurological problems often, but it is one of the most interesting subjects we have in medicine.

PLASTIC SURGERY

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(Presented before the New Mexico Medical Society, at its Fiftieth Annual Meeting, at Santa Fe, N. M., May 19-21, 1932.)

The subject of plastic surgery is so broad that only certain phases of it can be discussed. Today, I am discussing that phase which has to do with the transplantation and shifting of skin. This phase of plastic surgery constitutes its most important branch—in fact, in nearly all instances, one must shift skin or transplant it in some manner.

Some of the oldest operations known to the history of man have been of a plastic nature. The reconstruction of the amputated nose dates way back in history. In ancient India, the tile-makers caste reconstructed noses by bringing a flap down from the forehead and, from this procedure, we have the Indian method of flap transplantation. Many years later, namely in 1597, Gasper Tagliocozzi, an Italian, described a method of rhinoplasty by taking the skin from the arm, and from this we have the Italian method of pedicle graft, namely, skin taken from a distance.

In the early twelfth century in France, we have the first authentic record of free skingrafting. At that time, a woman charlatan in the streets of Paris was attemping to sell a salve. Unable to do it, she took a knife, cut a piece of skin out of her leg, put it back on the leg and pasted it over with salve. Several days later, she showed the populace that the

graft grew, and she sold a great deal of her salve, consequently.

With the continuation of wars and the increase of industry, plastic surgery has come more and more into its own. Nowadays, the automobile and industry are playing a greater part in the production of disfiguring injuries than even war.

When we pick up the text books, we notice that they talk of "autografting," or grafting from the same host; of "homografting," or grafting from another individual of like species; of "heterografting," or grafting from entirely different type of host.

Heterografting, that is, from unlike species—such as the grafting of frog skin and pig skin on humans—is impossible, unsurgical, and untrue. In all the literature of all the world, there has never been a successful case recorded that can be substantiated by the microscope. It merely acts as a covering under which the skin grows, either from the side or from islands which have remained in the denuded area. Likewise, it has been quite definitely established that one cannot grow skin from like species, even though the blood group is alike. This is true even though we use the skin from parent to child or child to parent. Lexor, Blair, McWilliams, Holden, and innumerable workers, have quite definitely proved that such grafts never reproduce a living cell and that the graft wholly disappears in a very short time. Those few cells which remain are highly questionable. If one were to pigment such a skin and transplant it, one would find that, in every instance, within two to four weeks, even though there is no infection, this whole graft would melt away like thin ice. Consequently, there is only one place to take skin, that is, from the same individual, and all other methods must be discarded.

I have mentioned this so emphatically because every once in a while one picks up a newspaper and reads that there has been a request made by a local surgeon for people to give their skin for burned children. This only subjects many people to unnecessary pain, the liability of infection, and a great deal of unnecessary trouble; and, in the certain disaster to the graft, we only decry good surgery.

There are two great divisions of skin transplantation: (1) true grafting, in which the skin is entirely separated from its bed and transplanted to a distant place; (2) where skin is shifted without entirely cutting it loose.

When using absolutely free grafts, we use four methods: (1) Fine pin-point-type grafts are occasionally used in badly infected, granulating areas. (2) We use very thin tissue-paper-like grafts, which are known as Thiersch grafts. (3) We use an intermediate type of graft which has been called a split graft. (4) We use a heavier type of graft; namely, the full-thickness graft.

The second graft, the very thin graft, is only used because it readily grows, since the area underneath it is almost certain to contract badly, causing the graft to crinkle.

The third method, the split graft, is the type of graft we most often use. With a suction machine, we are able to cut this graft in almost any desired thickness. Under ideal conditions, we can cut a graft as large as six by sixteen inches without trauma. It is easy to cut a graft six inches by ten inches. In order that these grafts may take, the grafted surface must be clean and free from infection, must be either freshly denuded or have a clean even granulating surface. The graft must be sewed in place and even, smooth pressure maintained for at least five days. We usually leave the dressing a week without changing it.

For the full thickness graft, one can, of course, cut it any size desired. It is seldom safe to cut this graft larger than six by eight inches. This graft is particularly adapted to the grafting of the palms and the fingers of burned children following contractures, and to replace certain contractures around the eyes and on the forehead. It is very good to cover elbow-joints, knee-caps, or any other area, such as in the popliteal spaces or axilla, in which we wish to have a rather large area of soft skin. The area to be grafted must be absolutely clean, must be smooth and regular; the graft should be sewed in place with just a slight amount of tension. It must be firmly and smoothly held in place and not disturbed for at least a week, and uniform pressure must be maintained for fully two weeks.

The second great group is the shifting of skin without completely severing it, that is, with the maintainance of blood supply from a pedicle. When we wish merely to close over an area by sliding sidewise, where the base of the flap is equal to or greater than the length, it is usually safe, if the blood supply is good, to shift this skin at one operation. Whenever there is a doubt, or whenever the length of the graft is three or four times the width of the base, one should always lift the graft and replace it in its original bed, allowing it to remain there about three weeks before attempting to shift it.

There are so many phases and so many different combinations of skingrafting, that I am only touching on some basic principles. In the first place, whenever possible, the flap should contain a major artery with an unimpaired venous return. Second, the field should be absolutely clean and free of infection. Third, before cutting the pedicle, the vitality of the graft should be tested by compression of the pedicle or gradually cutting it. It is seldom safe to cut the pedicle under two weeks and is seldom necessary to leave it longer than three weeks. Conditions, of course, must guide one. One must be careful, likewise, not to cut the flap too thin or to remove too much fat, as we know the small vessels that supply the skin run in the superficial fascia, namely, between the true skin and the fat.

When a very long graft is desired, one allows it to be attached on both ends and brings the graft together, making a tube. In that way, we prevent infection of the graft and then switch a very long flap. Likewise, if one wishes to get, for instance, a large smooth area in the neck and has no skin close by from which to take it, he can transplant a flap from the abdomen onto the arm, and then switch it

from the arm to the neck. This is called "jumping."

In conclusion, there is no sure way to success, but if the fundamental rules are followed, skin transplantation today is highly practical. With the exception of the transplantation of very large full-thickness grafts on uneven surfaces, we today can almost promise success. Our failures are very few and far between and, even with a transplant of a rather large area of full-thickness free graft, we seldom lose all the graft and are quite successful in the majority of cases.

DISCUSSION

DR. L. E. GRISWOLD, (Oklahoma City):—I just want to say a word in behalf of Dr. von Wedel's work. I have been working with him for the period of a year and, while I cannot contribute anything to plastic surgery, we are very proud of the doctor's success in Oklahoma City. He has twenty or thirty of these plastic cases on which he is working all the time, and the results are always excellent.

DR. W. H. WOOLSTON, (Albuquerque): This is a most interesting subject, both to the surgeon and to the laity, and Dr. von Wedel is to be congratulated on his excellent results. This is very tedious work; the after-treatment requires a great deal of care and patience. Dr. von Wedel has given us a very interesting and instructive paper, for which we are grateful.

FACIAL PAINS — DIFFERENTIAL DIAGNOSIS AND TREATMENT

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Since the studies of Sir Charles Bell (1829), to whom we are indebted for the knowledge that many of the facial neuralgias are due to disorders of the fifth nerve rather than the seventh nerve, great progress has been made. The neuralgias of the face are many and varied. Some are amenable to surgery; some yield to medical therapy; and to others, at present, we can afford no relief. One cannot too greatly deplore the many useless surgical procedures carried out for the relief of facial pain, such as the extraction of teeth, operations upon the nasal sinuses, intracranial section of the fifth cranial, sympathectomies, mastoid, sinus and even abdominal operations.

TIC DOULOUREUX.

Tic douloureux—also called trigeminal neuralgia, chronic paroxysmal neuralgia, epileptiform neuralgia, major neuralgia and Fothergills' disease—was known to early civilization. Trigeminal neuralgia was recognized by Avicenna in A. D. 1000, and was later described by Schlichtung (1748), Nicolous

Fig. 1. A composite sketch illustrating the position of needles for alcohol injection. (A) Extra-oral maxillary nerve injection (Levy and Baudouin). (B) Extra-oral maxillary nerve injection, (Schloesser). (C) Extra-oral mandibular nerve injection (Levy and Baudouin).

Andre (1756), who first named it "tic douloureux," and Fothergill (1773), who accurately described the disease.

The pain of tic douloureux is probably one of the most severe known to medicine. The pain occurs about equally in men and women, and the right side is more frequently involved than the left. The average age of onset is approximately fifty years, although cases have been reported at twelve years for the youngest and eighty-seven for the oldest. It is confined mainly to the second and third divisions, rarely to the first.

The first onset of pain is usually sudden and violent. The pain may then leave and the patient be pain-free for months, or even years. After the disease has once established itself, however, the patient has acute paroxysms of sharp, lancinating, knife-like pains, occurring in attacks of a few seconds, with intervals of freedom. The period of repeated attacks may last from several days to several weeks, and then a remission of months to years may occur. As the time from the original attack increases, the intervals of freedom are lessened, and the morale of the patient is decreased so that he is in constant fear of an impending attack.

The attacks are brought on by contact, and even the draught of air. Occasionally, the only relief from pain is by going to bed in a dark room and entirely abstaining from food. Trigger zones, or certain sensitive areas in the face upon which any slight pressure brings on an attack, are characteristic of the disease.

The pain of trigeminal neuralgia is always confined to the sensory distribution of the fifth nerve, never going beyond its bounds. The pain is superficial and there is an absolute freedom from pain between these attacks, although occasionally there is a discomfort present in this interval. It is a unilateral disease, primarily, though there have been reported in the literature some fifty-four patients with bilateral pain. Trigeminal neuralgia is a disease of unknown etiology and no single case has ever been cured by the clearing up of oral sepsis, the extraction of carious teeth, the treatment of apical abscesses, or sinus operations. In addition, no single case has ever had a permanent relief of pain without therapy.

Treatment: Trichlorethylene is of value for the relief of pain in approximately 11 per cent of the cases. This drug was discovered by chance in Germany during the World War. The dosage of this drug is as follows: inhalation three to four times a day, from 20 to 25 drops being placed on a piece of gauze. The inhalation should be continued until the odor has entirely disappeared. This should be carried out over a period of a month to six weeks before its use is discontinued. The drug occasionally produces vertigo, sleep, and drowsiness, and therefore it is well to take the inhalation in a recumbent position. After the pain is relieved, it is not necessary to continue the inhalation of the drug daily. As a prophylactic measure, it is extremely wise to inhale this drug every two or three months for a period of three consecutive days. When the patient is relieved by trichlorethylene therapy, sensory anesthesia does not occur.

Alcohol injection of the second and third divisions is only palliative, the relief usually occurring from six to eighteen months, occasionally as long as seven years. The unfortunate fact is that, after repeated alcohol injections, a period arises wherein further injections of the nerve do not relieve the pain, primarily because a marked fibrosis has occurred about the nerve, thus preventing the infiltration of alcohol. In Figure 1 the methods of alcohol injections are indicated as used by the neurosurgeon, though the usual mandibular block utilized by the dental surgeon is efficacious for third-division pain. Alcohol should never be injected into the first division, because of the proximity of the nerves to the eye and the resulting dangers frequently arising therefrom, particularly paralysis of the ocular muscles, and occasionally blindness. I, personally, do not favor alcohol injection of the ganglion, because of the danger of infiltration into the pons along the nerve branch. Numerous such cases of pontile destruction have been reported in the literature.

The surgery of the trigeminal tract has reached the stage of great efficiency. The pain can be absolutely relieved permanently with the minimum risk to the patient, with the surprisingly low mortality of 0.26 per cent. The present accepted operation is that described by Spiller and Frazier, wherein the sensory root is partially sectioned. As the disease rarely involves the ophthalmic division, the sensory supply to the cornea is maintained so as to prevent a keratitis (fig. 2). The operation can be performed as readily in the old as in the young, and leaves the patient without any facial disfigurement. Intracranial section of the three divisions of the trigeminal nerve, as well as removal of the ganglion, has given way to subtotal section of the sensory root. Conservation of the motor root is also carried out so as to prevent

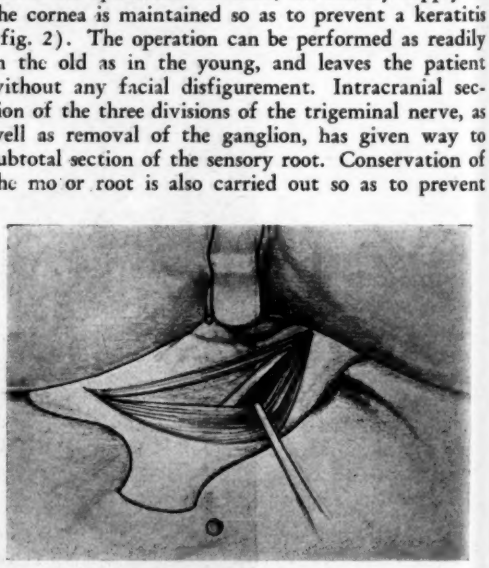

Fig 2. Subtotal resection of the trigeminal nerve. The lower one-third of the ganglion has been retracted, showing the motor root in situ. The upper one-third of the ganglion intact, so that the sensory supply to the cornea is preserved thus preventing a keratitis.

paralysis of the muscles of mastication and to leave the patient in a position for a bilateral operation, if such may be needed (fig. 3).

Fig. 3. Case wherein a subtotal resection of the sensory root has been performed. Note the absence of scar, as well as absence of facial paralysis.

SECONDARY TRIGEMINAL NEURALGIA.

POST HERPETIC TRIGEMINAL NEURALGIA

During the acute attack of herpes, a severe, continuous pain is referable to the trigeminal distribution and usually disappears with the healing of the cutaneous lesion. Occasionally the pain may persist for several weeks and then gradually subside, and still more rarely, the pain may continue on for years without cessation, varying in intensity and closely simulating the pain of true trigeminal neuralgia. Very often all therapy directed to the trigeminal tract, even with section of the sensory root, does not give relief. This is true particularly in the aged and the complaint is of a constant, dull, burning and aching pain, accentuated with paroxysms of the sharp, shooting variety. This can only be explained on a basis of central pathology, such as a perivascular infiltration in the internal capsule. These cases must be approached with great skepticism, considering the probability of central pathology accounting for the pain, rather than any involvement of the peripheral tracts of the trigeminal nerve.

TUMORS OF THE GASSERIAN GANGLION

Tumors of the Gasserian ganglion result in pain referred to the trigeminal distribution. However, as a tumor is definitely invasive and destructive, pain is a manifestation of irritation, whereas the numbness which occurs in these cases is caused by nerve destruction. The numbness is diagnostic of tumor. Frequently the earliest clinical sign manifested is an absence of the corneal reflex, and, should this exist, the diagnosis of tumor is most likely. Tumors may occur primarily in the ganglion, the sensory root, or may involve the ganglion secondarily from other regions in the brain (fig. 4).

INFLAMMATIONS OF THE GASSERIAN GANGLION.

Inflammations of the Gasserian ganglion are extremely rare, and may be secondary to an otitis media with abscess formation of the ganglion. Gradenigo's syndrome, which consists of otitis media, with or without involvement of the mastoid, is accompanied by sixth nerve palsy as well as trigeminal pain. The production of this trigeminal pain has not been definitely explained. Gradenigo attributed it to infection in the air cells at the tips of the petrous portion of the temporal bone, and also in the region of the ganglion, as it lies over these cells. Vogel considered it

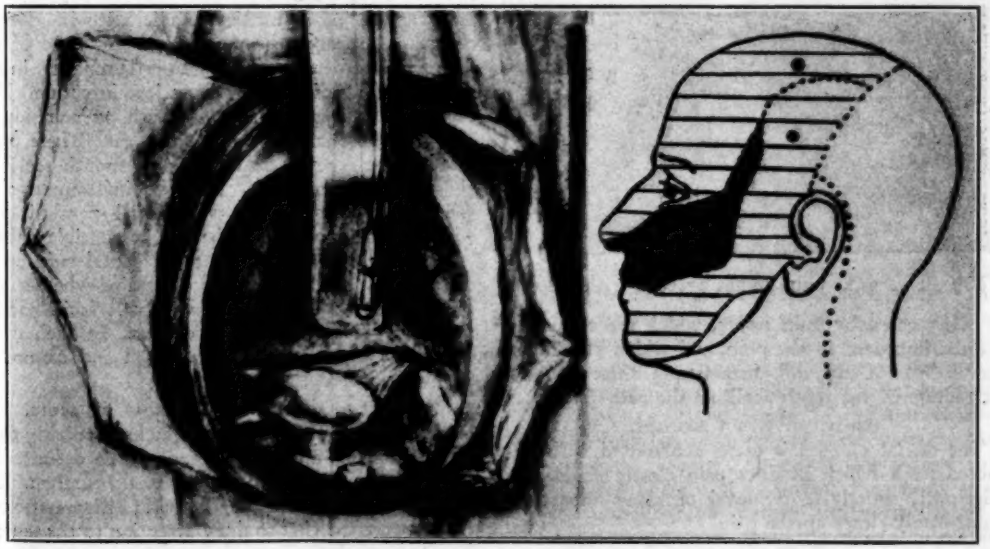

Fig. 4. A tumor of the Gasserian ganglion. The adjoining figure shows the area of sensory anesthesia.

due to a localized meningitis over the apex of the petrous bone, and Kuick believed it due to a toxic neuritis.

ANEURISMS OF THE INTERNAL CAROTID.

Aneurisms arising from the internal carotid, or in portions of the circle of Willis, may reach such size as to press upon the ganglion itself, or upon the branches of the fifth nerve. Pain referred to the trigeminal distribution may be the first sign of cerebral aneurism, anesthesia developing at a later period.

MULTIPLE SCLEROSIS.

Multiple sclerosis may occasionally manifest itself by pain in the trigeminal distribution. Of course, other neurological signs are necessary to clinch the diagnosis and are always present.

SYPHILIS.

Syphilis may occasionally involve the Gasserian ganglion and produce symptoms of trigeminal neuralgia. For this reason, it is well to take a Wassermann in suspected cases.

THROMBOTIC LESIONS.

Thrombosis of the posterior inferior cerebellar artery may result in degeneration of the fifth root of the fifth nerve. Pain would then be referred to the trigeminal distribution. Here, again, additional neurological signs will definitely rule out true trigeminal neuralgia.

DENTAL PULP STONES.

A condition causing severe attacks of lancinating pain referable to one tooth, or several teeth, is a disease seen more frequently by the dentist. It is caused,

Fig. 5. Dental pulp stones present within teeth.

in many cases, by pulp stones, which are calcareous nodules imbedded in the pulp, and which press upon the nerves. X-ray will demonstrate these nodules. Extraction of the tooth abolishes the pain (fig. 5).

IMPACTED THIRD MOLARS.

The dental surgeon is often confronted with impacted teeth which produce pain closely simulating trigeminal neuralgia. Removal of these impactions immediately clears up the pain.

TRAUMA

Following fracture of the skull, as well as fracture of the jaw, the ganglion may be invloved, as well as the peripheral portion of the trigeminal tract. The pain here usually clears up following regeneration of the nerve.

UNKNOWN ETIOLOGY

Occasionally pain referable to the trigeminal tract may be continuously troublesome—though not excruciating. Possibly a distant focus of infection may be the etiological factor, but of this one cannot be too certain. Following an alcohol injection, the pain instantly subsides and, when the alcohol wears off, the pain does not return.

NEURALGIA DUE TO FACIAL MALIGNANCY

Malignant disease about the face and neck, with the terrific pains that result therefrom and the extreme discomfort associated with sloughing surfaces, make the patient decidedly miserable. The cauterization and x-ray treatment, which is carried out, causes the most torturing pain. It is in these cases that injection of alcohol, or section of the various nerve roots, will greatly ameliorate pain and lessen the patient's suffering and will, in addition, permit the surgical and plastic procedures to be performed painlessly.

Pain deep in the ear is a symptom difficult to relieve, and it is for this reason that section of the glossopharyngeal, or the tenth nerve, may be indicated. Upon rendering these patients pain-free, the morale is greatly increased, morphine unnecessary, and even though these patients realize that the procedure has nothing to do with a cure of their primary disease, they are deeply grateful for the relief of this continuous, terrific, and unbearable pain.

SEVENTH CRANIAL NERVE

The sensory supply to the ear is unusually complex and has been attributed to the fifth, seventh, eighth, ninth and tenth cranial nerves and the cervical nerves. Thus we note ear pain may be produced by involvement of numerous cranial nerves. Reflex otalgia is probably the more important type of ear pain which is often secondary to inflammation in the mouth and to diseased teeth or jaw infection. Not infrequently does an infected tooth on one side of the jaw refer pain to the opposite ear. These pains will not be relieved by clearing up mouth sepsis or the extraction of teeth. Herpetic inflammation of the geniculate ganglion, with facial paralysis and acoustic symptoms, may be the responsible factor. Therefore, it is advisable for the dental surgeon to examine the ear carefully to determine whether any small vesicles are present, or whether a facial paralysis exists, with or without deafness. In such cases, extraction of teeth is without avail and cannot in any way relieve the pain.

Idiopathic otalgia, which consists of acute, paroxysmal lancinating pains, in attacks occurring daily or with longer intervals of freedom, is a definite disease entity very much like trigeminal neuralgia. The studies of Ramsay Hunt on herpetic inflammation of the ear were the first to draw attention to the sensory distribution of the seventh cranial nerve to the ear. Later, Taylor and Clark reported a definite case of seventh nerve otalgia, which was relieved by section

of the seventh nerve and the nerve of Wrisberg, as well as part of the eighth nerve. This type of neuralgia is extremely rare and the author has had only one case under his observation. This was a young man, aged 38, who for the past four years had had attacks of terrific, lancinating, burning pain situated in the tragus, antitragus, and lobule of the left ear. These attacks would occur rather insidiously; the ear would throb, become red, and gradually, after several hours, the pain would reach an unbearable height. When it seemed almost impossible further to endure this pain, it would suddenly cease—very much as if an abscess under tension had been incised. For some six to eight hours after, a feeling of discomfort would persist in the ear. As the attacks occurred only at lengthy intervals, and in view of the fact that there had been only three attacks in the four years, surgery was deferred until the attacks became more frequent.

NEURALGIA OF THE EIGHTH CRANIAL NERVE.

Ringing in the ear, associated with deafness, may frequently be considered secondary to a dental involvement. However, there is a definite neurological syndrome, Meniere's disease, which consists of attacks of nausea and vomiting, with ringing in the ears and deafness. This is caused by pathology of the eighth nerve. At times these attacks may become so severe that the patient is completely incapacitated. At other times, the interval of freedom is quite lengthy and the patient may be up and about most of the time. If the attacks are of great severity, section of the eighth nerve, as described by Dandy, absolutely relieves this clinical picture. Persistent ringing in the ear should always be looked upon with suspicion as many times tumors of the eighth nerve manifest themselves at an early date by this important clinical symptom.

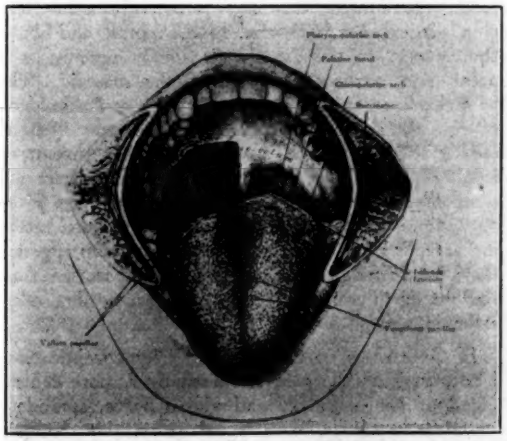

Fig. 6. Sensory supply of the glossopharyngeal nerve. This indicates the area of pain.

NEURALGIA OF THE NINTH CRANIAL NERVE.

The sensory supply of the glossopharyngeal nerve may be noted in figure 6. That this pain may be confused with pathology referable to the mouth, or oral sepsis of the mouth, or diseased teeth, may readily be comprehended. The pain in glossopharyngeal neuralgia is acute, spasmodic, lancinating and occurring in attacks lasting a few minutes. The pain starts in the pharynx, the tonsillar fossa, or the base of the tongue, and radiates to the ear, the neck, or the tympanum. The trigger zone is usually in the tonsillar fossa. This pain may be brought on by swallowing or yawning. Relief may be readily obtained by intracranial scetion of the glossopharyngeal nerve.

NEURALGIA OF THE TENTH CRANIAL NERVE.

Occasionally, deep-seated pain in the ear and throat, particularly due to invasion of the face by carcinoma, may be aided by section of the tenth nerve. The evidence for this point, however, is extremely scant and much more work must be carried out to justify such surgery. Pain of tuberculous laryngitis is referred to the superior laryngeal nerve of the vagus and can be relieved by injection of alcohol or by section. This pain is brought on by swallowing because of irritation to the larynx and epiglottis by food passing over the structures.

ATYPICAL FACIAL NEURALGIA.

This neuralgia is one of the most important, primarily because of its unknown etiology and the failure of clinical relief. As the physician is unable to alleviate the pain, the patient is then referred to the dentist for the extraction of teeth. Unfortunately, this does not relieve the patient from his suffering. More frequently the pain becomes worse, and in addition to complaining of a more severe pain, the patient is minus his or her teeth. Practically all patients referred to the author with atypical neuralgia, have had one or more teeth extracted, and all of the patients declared their pain had been increased by this procedure.

From the group of trigeminal neuralgias have been separated a series of patients complaining of continuous facial pain, which is not relieved by section of the sensory root, or any other procedures which relieve the pain of trigeminal neuralgia. After a more meticulous study of patients suffering with this pain, it was found that the distribution was not in the trigeminal tract, but continued beyond its bounds. This pain may locate in single areas (fig. 7), or in an entire arc (fig. 8).

The pain of atypical neuralgia is persistent and continuous. There are, however, at intervals of several days to several weeks, severe exacerbations of pain which last from two or three hours, to three or four days. During this period the patient may be bedridden and suffer intensely. Frequently these severe attacks begin rather insidiously, reach their height after two or three hours, and then, possibly at the end of the second or third day, slowly subside so as to resume the chronic stage. During this

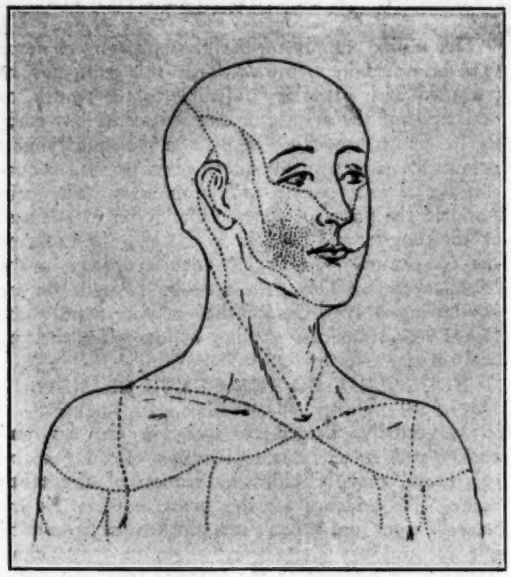

Fig. 7. A single area of pain of the atypical type.

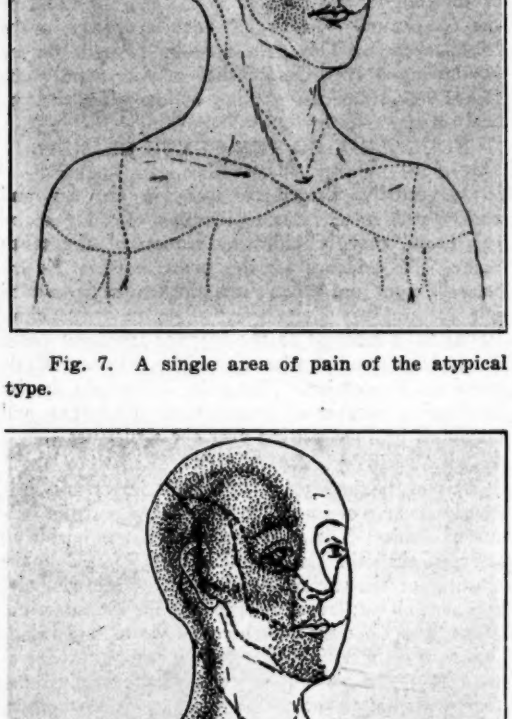

Fig. 8. A complete arc distribution in a case of atypical neuralgia. Note how the area of pain crosses the broders of the trigeminal distribution.

chronic phase, the patient will complain of persistent pain, yet there is no evidence of pain in the facial expressions. These patients may talk to you, explain their symptoms and, when questioned as to the presence of pain at that particular time, will always say that they are suffering with pain at that moment. This is quite different from trigeminal neuralgia, wherein the attacks last only a few seconds, are excruciating, but the patient is entirely pain-free during the intervals.

In trigeminal neuralgia, the pain is on the surface of the face, because it is caused by the trigeminal nerves: in atypical facial neuralgia, the pain is deep-seated, because it is probably the result of a disease of the sympathetics of the blood vessel, and it, indeed, follows the outline of the arteries. The pain is extremely difficult to describe and an extraordinary number of adjectives have been utilized by patients. Associated with this pain are sympathetic phenomena, such as lacrimation, edema of the eye lids, unequal pupils, corneal injection, exophthalmos, salivation, nasal discharge, flushing of the face, aural discharge, nausea and vomiting, perspiration.

Many procedures have been utilized for the relief of this pain, all, however, proving of little avail. Among the procedures attempted for the alleviation of this pain have been: injection of alcohol in branches of the trigeminal nerve; cocainization and injection of the sphenopalatine ganglion; extraction of teeth; drainage of sinuses; supra-orbital and infra-orbital nerve avulsions; nasal operations; cervical sympathectomy (Frazier); stripping of the peri-arterial plexus; subtotal section of the sensory root of the trigeminal nerve; mastoid operations, and pelvic operations. Of late, a realm of hope has been given these sufferers. Paravertebral block has relieved an occasional case, and an occasional report of relief by section of the stellate ganglion appears in the literature. Upon a clearer comprehension of the sympathetic system, it is quite probable that these unfortunate patients may eventually be relieved from pain.

SPHENOPALATINE NEURALGIA.

Sluder, after a careful study of the anatomical relationship of the sphenoid and posterior ethmoids, demonstrated that in many cases these cells were in close proximity to the nasal ganglion. He assumed that, if inflammation of the optic nerve could occur from these sinuses, there was no reason why the nasal ganglion would not suffer.

A history of coryza is followed by pain in the root of the nose, in and about the eye, the upper jaw and teeth, occasionally the lower jaw and teeth. This pain also extends backward to the temple and above the zygoma to the ear, and is always severest at a point five centimeters back of the mastoid. The pain also radiates to the occiput, neck and shoulders, or even the arm, forearm, hand and fingers. Associated with this pain is a stiff or aching throat, or itching of the hard palate. In addition, there are sympathetic symptoms which may occur even without pain. The patient is seized with severe sneezing attacks and a thin, hot profuse secretion appears; the eyes are reddened; there is increased lacrimation; the pupils dilate and there are dyspnea, dry rales, asthenia, and photophobia.

If these cases do not get better by cocainization, Sluder believes the pain is caused by a more central lesion of the maxillary and vidian nerve, secondary to sphenoidal inflammation. Intrasphenoidal application of cocaine is then indicated. Injection of the ganglion, or applications of formaldehyde and silver are indicated in the more severe cases. If the pain is

not relieved by these treatments, the spenoid should be operated upon, because the nerve and ganglion would then be imbedded in a chronic, inflammatory tissue. Many of these cases have only a transitory relief. These cases Sluder could not explain clinically and believed future study would necessarily have to solve the problem. As this pain frequently is diversified, teeth also are extracted. It has been the author's experience that the chronic picture of sphenopalatine neuralgia does not exist; that is, patients suffering from pain lasting for a period of six months or more, usually belong to the atypical neuralgia group; whereas, the very acute pains, seen early, and of short duration may be relieved by cocainization. Frequently, acute sphenoiditis produces the very symptom picture described above and relief is brought about by drainage and cocainization of the sphenopalatine ganglion.

PAINFUL TIC CONVULSIF.

This clinical picture was first described by Cushing. It consists of a spasmodic contraction of the facial muscles, accompanied by intense pain. All types of surgery were carried out for this picture, such as: the extraction of teeth; surgery of the trigeminal tract; section of the facial nerve. None, however, resulted in any relief of pain or muscle spasm. Whether this disease is of central origin is not known, but it may also be classified among the incurable types of pain.

CONCLUSIONS

Surgery offers a relief for many of the neuralgias of the face, with an exceedingly low mortality of 0.26 per cent.

Atypical neuralgia and painful tic convulsif are types of pain which should be recognized so that useless surgical procedures may not be attempted. At present, we can offer no definite relief for these types.

Neuralgias of the face very frequently are secondary to systemic diseases, which requires a complete medical examination of such patients, as well as dental study.

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BRAIN ABSCESS FROM THE OTOLOGIST'S VIEWPOINT

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(Presented before the New Mexico Medical Society, at its Fiftieth Annual Meeting, held at Santa Fe, N. M., May 19-21, 1932.)

This paper is written to call your attention to the status of brain abscesses as seen by the average physician. I shall first present for your consideration cases taken from the records of Saint Anthony Hospital and Northwest Texas Hospital, Amarillo, Texas. These cases were seen and diagnosed by many different types of practitioners. Outside of three or four, no attempt was made to diagnose the site of the lesion or to institute treatment other than palliative, the reason being the hopelessness of the cases when seen and the realization that treatment in the later stages is futile. The records indicate two things: (1) the inclination to procrastination on the part of the patients and the hopelessness of the condition

during the late stages; (2) the advantage that could be had from having trained neurologists and brain surgeons in all hospitals. I call your attention to these records not as a critic, but to draw conclusions for our mutual benefit.

CASE HISTORIES

Case No. 1623. St. Anthony Hospital record. White male, age 41 years. Entered hospital October 18, 1931. History of right ear discharging since childhood. Complains of severe pain in right temporal region, of one week's duration, word aphasia, and tenderness over right mastoid region. No rigidity of neck. Respiration 20 to 35; pulse, 60 to 90; temperature, 100.5° to 104.5°. Blood count: W. C., 13,000; poly, 90; spinal cell, 12 cm. Wassermann negative. Operative record shows cholesteatomatous material in antrum, pus was found in the temporal lobe and right ventricle. Patient died October 26, 1931.

Case No. 895. St. Anthony Hospital record. White male, age 54 years. Entered hospital June 12, 1930. History of several Neisserian infections. Wassermann negative. Symptoms began about two months ago with projectile vomiting. One week ago, vomiting occurred spontaneously and with headaches. Choked disk, left pupil elliptic, does not react to light and distance. Right pupil reacts sluggishly. Exaggeration of deep reflexes. X-ray does not show demonstrable lesion of head. Answers questions slowly but correctly. Spinal fluid came out under pressure. Cell count, 20. Blood count: W. C. 20, 600. Urine, 3 plus albumin. Temperature ran from 97° to 96°; pulse, 70 to 90; respiration, 15 to 20. This patient died June 18, 1930.

Case No. 1738. St. Anthony Hospital record. Negro boy, age 15 years. Entered hospital November 5, 1930. Was admitted to hospital in an unconscious condition. History of discharging left ear for about one week previous to entering hospital. Suddenly felt a severe pain in left side of head, no rigidity of neck, but paralysis of left side of face. Blood count: W. C., 28,400; poly, 92; small lymph., 16; large lymph., 2. Diagnosis brain abscess. Died six hours after admission to hospital.

Case No. 2334. St. Anthony Hospital record. White girl, age 7 years. Eight or nine days previous to entering hospital on May 7, 1929, she was seized with a severe pain in the head, following a chronic ear discharge, right ear. Had continued high fever—104 degrees,—profuse sweating and chills. Temperature previous to death, 107 degrees; pulse, 150; and respiration, 38. Died six hours after entering hospital.

Case No. 454 St. Anthony Hospital record. White female, widow, age 26 years. Admitted to hospital on March 24, 1930, 4 p. m., in an unconscious condition, and died 12:07 a. m., March 25, 1930. The history, which was given by her parents, was of an ear infection of many months' duration. Suddenly became unconscious on March 24, 1930. Temperature, 106°; pulse, 140; respiration, 10. Wassermann positive (3 plus). Spinal fluid cell count, 2 per cm. Blood count: W. C., 19,550; poly., 93; small lymph., 5; large lymph., 2.

Case No. 8945. Northewest Texas Hospital record. White male, age 51 years. Family history negative. Habits good. Has been sick for eighteen months, slight headaches every two or three days. For past two days has been nauseated, vomiting, extremely nervous, and picking at bed clothes. Entered hospital February 7, 1930. Temperature on admission, 98.6°; respiration 24; blood pressure, 118/60. There was a paralysis of left extremities, no reflex on left side. History of chronic running ear for ten years, right side. Pus from ear very foul. Blood count: W. C., 16,000; poly., 88. Died February 10, 1930. Postmortem: The calvarium was removed in the usual manner and the brain exposed. Then the dura was dissected off. The pia mater was intensely injected and adherent. A slaty-gray area, 2.5 cm. by 3 cm. in diameter, with a small sinus leading from the third ventricle to upper portion of mastoid area and connecting with antrum, filled with pus, was found.

Brain abscess is a common complication of middle-ear disease. It may occur during an acute process, but more frequently follows during the course of a chronic middle-ear suppuration. The otologist meets with brain abscess only occasionally, but sufficiently often to impress him with the seriousness of the condition. It is a condition requiring surgical intervention if the patient is to live. The mortality, even with operation, is frightful, but it is the only hope and death is almost sure and certain without it.

The symptoms of brain abscess are often vague and ill defined. Even when present, it is hard to differentiate from meningitis, and when diagnosed, the site of the lesion is often extremely difficult to determine, but we have certain signs which, if followed, will lead us, at least, to a fairly accurate knowledge of conditions present. These signs are:

1. Any inflammatory process of the middle ear, acute or chronic, is a potential focus for brain abscess. If the middle ear is infected, the route is short to the brain substance, so, in the presence of a discharging ear, if there is sudden pain, or a chill, or any symptom uncommonly associated with the discharge, our attention should immediately be on guard as to brain complications. Patients with chronic running ears give premonitory signs of impending danger by not feeling well, malaise, restlessness, indigestion, loss of weight, weakness of arms and legs, and headaches. It is said by some observers that brain abscess can be latent for many years before development of acute symptoms.

2. Headache: There is headache, severe head-

ache, unilateral mostly, diffuse at times. No drug gives relief, except very temporarily. There is sometimes a tendency for the headache to diminish. This may be due to the dulling of perception as the result of increased intracranial tension. A periodicity is noted in some cases.

3. Mentality: Case No. 895, St. Anthony Hospital records show, "Patient answers questions slowly but correctly." This is typical. A question is asked, the patient acts as if he had not heard, then answers slowly. Reasoning is slow and difficult, ability to concentrate is lost. Drowsiness is frequently seen. The patient is apparently asleep and has to be aroused for the administration of food and drugs. This drowsiness develops into coma and death. If the abscess ruptures into the ventricles, death may occur more suddenly.

4. Pulse: At first the pulse is rapid. It gradually becomes slower, but just prior to death it becomes much accelerated.

5. Ocular signs: Ocular signs must be in rela tion to the findings. To base conclusions on eye findings alone will lead to many errors. There is often no change in the eye grounds. When there is, there is a variation from a slight haziness of the optic disk, through all the grades of optic neuritis to choked disk. Heretofore, much delay in diagnosis was due to a misconception of the choked disk. It is now commonly recognized and appreciated that a measurable papilledema is the end result of a continued pre-existing intracranial pressure and that dilated retinal veins and an edematous swelling at the margins of the optic disk are the early signs. No longer is it necessary to wait for a measurable papilledema before a diagnosis of intracranial pressure is made. Eye-ground changes may be found in either or both eyes. When unilateral, the changes are not always on the side of the lesion. The factor in optic neuritis development depends to a degree on the local condition within the eyeball. It develops in the eye that has the lower intra-ocular tension, regardless of the site of the abscess. The changes in the field of vision are helpful but not conclusive. Nystagmus is present or absent according to the site of the lesion. The direction is often hard to determine, so we often confuse ourselves by trying to determine the direction. It is a symptom, however, that should be taken into consideration.

6. Temperature changes: The temperature varies. Typically it is moderately high at first, Then low, and, near death, is extremely high. Chills are sometimes seen, but profuse sweating is rare.

7. Blood picture: There is no typical blood picture. The leukocyte count is from 12,000 to 20,000. The polymorphelar leukocyte percentage is high.

8. Vomiting: Many of these patients vomit. When first seen the symptoms may lead one to think of some gastro-intestinal condition. When a patient has a running ear and vomits, the ear, and not the stomach, should receive first consideration. A convulsion occurring during the course of a discharging ear is almost pathognomonic of a brain abscess.

When the abscess affects the cerebellum, which is the coordinating center of the brain, there is a disturbance of muscle movements, manifested by unsteady gait and a tendency to fall. This is an early symptom and progresses to a paralysis in the late cases.

9. The x-ray does not often reveal an abscess of the brain and is of little aid.

10. Spinal cell count: So great an authority as W. P. Eagleton, in a paper before the American Academy of Opthalmology and Otolaryngology, once said, "I never do a lumbar puncture in any case where there is even a suspicion of an abscess which may involve either the posterior fossa or any part of the mid-brain cone that passes up through the tentorial ring." In these cases which I report, the attending physicians were less timorous, so we are able to learn how the spinal count runs in the majority of cases.

In the cases recorded here, the diagnosis was made too late to be of any benefit to the patient. They show the value of early diagnosis if surgery is to do anything other than hasten the end. Some one has very aptly said that "when a diagnosis is made of brain abscess it is too late to operate." The symptoms must be interpreted early if the patients are to be benefited. The classical symptoms cannot be waited for. When they appear, the case has progressed to a dangerous stage. It is important that we keep in our minds the potential danger of a chronic discharging ear, so that, when there is any deviation from its normal course—such as headache, vomiting, loss of smell, muscular weakness and other symptoms of this type—we be on our guard. Early diagnosis and prompt treatment are essential if we are to aid the patient.

DISCUSSION

DR. J. P. KANES (Santa Fe, N. M.): I am very appreciative of Dr. Duncan's splendid paper and think he deserves a great deal of credit for the intensive study he has made of this subject. In this connection, I am reminded of the immense amount of study and work done by Sir James Mac-

kenzie on the heart and how much the profession is indebted to him. Dr. Duncan is pursuing similar lines in his study of the brain and has brought to us, in my opinion, one of the most interesting papers of the meeting.

DR. DUNCAN (closing): I very much appreciate the pleasure and honor you have bestowed upon me by allowing me to appear before you. Like Mackenzie, I am a Scotchman, and can appreciate all the jokes perpetrated on my suffering kinsmen—for we all know that a Scotchman makes money first to make it last. I thank you very much.

IMMUNIZATION AND TREATMENT OF DIPHTHERIA

C. F. MILLIGAN, M. D.
Clayton, N. M.

(Read before the New Mexico Medical Society, at its Fiftieth Annual Meeting, at Santa Fe, N. M., May 19-21, 1932.)

Our knowledge of diphtheria dates back to the time of Hippocrates, and as early as 100 B. C. the clinical picture was pretty well known and laryngotomy was practised for its treatment; but not until early in the eighteenth century did the study of the disease become productive. The later history of diphtheria contains its two most important epochs: the discovery of the bacillus by Klebs in 1883 and its isolation and cultivation by Loeffler in 1884; and the discovery of antitoxin by von Behring in 1892. In recent years immunity against the disease has been established by the use of toxin-antitoxin and toxoid.

With a knowledge of the disease dating back over 2000 years and with a specific immunizing agent and a specific treatment, the death rate holds about fifth place of all infectious diseases of childhood. Too many physicians consider the diagnosis of diphtheria easy and the treatment simple, and this attitude, I believe, is largely responsible for the prevalence of the disease and the complications that we so frequently encounter. If the medical profession would awaken to its responsibility we could do for diphtheria what we have done for typhoid fever.

It is not the purpose of this paper to go into the subject in an exhaustive and time-consuming manner, but to touch a few of the important and practical points in the prevention and treatment, and thereby stimulate more interest in the prevention and the proper management of the disease.

Certain clinical and laboratory facts regarding diphtheria toxin are essential to a clear understanding of the disease and its treatment. Toxin is the name given to the substance produced by the development or growth of the Klebs-Loeffler bacillus, and it is this toxin that unites with the tissues of the body to produce the symptoms that we recognize in patients affected with the disease. The following facts should be noted: (1) The toxin is absorbed rapidly by the tissues and from there it reaches the blood stream. (2) It is not secreted in any appreciable amounts into the cavity of the throat and cannot be washed out by gargles. (3) The quantity of toxin produced by a case cannot be measured, but the severity of the local inflammation, and the toxic after effects are crude measures; the amount of anti-toxin necessary to save the patient is of value but the need of a margin of safety prevents this being accurate. (4) The toxin probably combines with many tissues of the body but only in a few, such as the nerves, the heart, and other organs, are harmful clinical effects found. (5) The absorbed toxin acts with great rapidity on the tissues so that a fatal effect may be produced within thirty-six hours. (6) It has not been shown that toxin-tissue combinations may be broken by antitoxin or other treatment, but when antitoxin is present in the blood stream the toxin always combines with it rather than with the tissues. (7) In susceptible animals, toxin leaves the blood stream with great rapidity, while in non-susceptible animals it may remain in the blood for a long period.

Natural immunity to diphtheria is quite common. Over fifty per cent of the population is immune. Acquired immunity from an attack of the disease may last for years or it may last only a few weeks. Artificial immunity is also variable.

Diphtheria is much less contagious than measles or smallpox. In fact, it requires fairly intimate contact to transmit the organisms from one person to the vulnerable points of another. The nasal form is considered the most contagious, and this form very often escapes detection, because it is mistaken for a bad cold. The laryngeal type is probably the least contagious. The duration of the contagion varies. It may last for only a few days, while, in the case of some carriers, it may last for several years.

Children under six months of age are not very susceptible to diphtheria. This is probably due to the transmission of antitoxin from the mother to the child through the blood in utero. This immunity usually lasts about six months and is probably gone by the time the child reaches one year of age. After this age the child begins to build up some natural immunity, which increases until about the age of

twelve. The Schick test is unreliable in children under six months of age because of the lack of reactivity of the skin.

Antitoxin may develop spontaneously without the stimulus of the diphtheria bacillus. The ability to develop antitoxin is largely dependent upon heredity factors. There are individuals and families who seem to lack this protective force and in these strains the disease is apt to run a malignant course.

Diphtheria has a tendency to occur periodically in communities where immunization is not practised. Active immunization has proved successful and should be a requirement for all school children, as smallpox vaccination is here in New Mexico.

At present, there are four preparations used in immunization against diphtheria. The most popular is probably the toxin-antitoxin, because it has been in use the longest and is better known. Toxoid is rapidly taking the place of toxin-antitoxin with many pediatricians. Anatoxin and Lowenstein's diphtheria salve are used quite extensively in some hospitals in Europe. Time will not permit me to describe the preparation of these agents. I have used the toxin-antitoxin and the toxoid and I prefer the toxoid to the toxin-antitoxin, because it is reported to have no reaction, it confers a higher degree of immunity in a shorter period of time and does not sensitize the patient to animal serum. The toxoid is said to confer an immunity of at least 95 per cent within six weeks after the completion of the immunization, and it probably lasts throughout the susceptible period of life. Bela Schick writes that the problem of active immunization to diphtheria is practically solved. If every precaution is observed there is no danger in its application.

There have been some sad experiences with the use of toxin-antitoxin. The first series of accidents occurred in Dallas, Texas, in 1919. Forty children who had been inoculaetd fell ill, five of them died. The mixture proved to contain too much toxin and little or no antitoxin. The second series was in Massachusetts in 1924 and was caused by a preparation that had been frozen. Since that time no accidents have occurred in America because steps have been taken to remedy the defects.

I wish that we had the time to go into the diagnosis of diphtheria, for I consider the diagnosis very important. Don't wait for the appearance of membrane before suspecting diphtheria. One may be compelled to make a tentative diagnosis by exclusion. Then give antitoxin and clear up the diagnosis afterward.

In the treatment of diphtheria, we first think of antitoxin, which is really the one and only treatment. The earlier it is given, the smaller the dose required and the better the results obtained. Abt believes that 1000 units administered on the first day is worth more than 10,000 units after the fifth day of the disease.

Antitoxin does not destroy the bacilli. It neutralizes the toxin produced by the bacilli; but after the toxin has united with the tissues the antitoxin has no power to break this union. Therefore, the damage, once done, is beyond the power of medication to relieve. All that we can expect from antitoxin is to neutralize the free toxin in the blood and prevent further tissue union. The size of the dose must therefore be determined by the severity of the case and the time elapsing before treatment is begun. Place states that no harm can arise from an overdose of antitoxin, but great harm may come from too small a dose. Abt thinks that we should give from 15,000 to 60,000 units, that from 10,000 to 20,000 units should be given intravenously and 30,000 to 40,000 units intramuscularly in severe cases. He believes that repeated doses may lead to gangrene and, if given as long as three or four days after the first dose, there is great danger of anaphylactic shock.

Children with diphtheria may die from either of two causes. They die from mechanical obstruction with membrane in the larynx or lungs, or they die from the effect of diphtheria toxins upon the various organs of the body. The first cause of death is easily recognized, but the latter is more remote and the death is attributed, in many instances, to conditions which have no connection with diphtheria.

Diphtheria begins as a localized disease, usually in the throat, and later becomes more general in its action. The toxin is a slow poison, taking some weeks to produce all of its effects.

Too many of us in general practice expect antitoxin to carry the whole load in these cases. As soon as we make a clinical diagnosis of diphtheria, we give sufficient antitoxin to bring about a reduction in temperature and alleviation of the other symptoms, and consider our patient on the high road to recovery and take his name off our visiting list. If, after a week or ten days, our little patient suddenly dies, or, a little later on, we are called back to see a case of paralysis, the relatives are sure that the complication was caused by the antitoxin, and we have a suspicion that the antitoxin was not as pure the manufacturers claimed it to be. We expected the antitoxin to cure our patient. Antitoxin in sufficient

doses neutralizes any toxins in the circulation and any that may be produced after its administration, but it will not neutralize the toxins that have been absorbed and become fixed in the tissues. If this be the case, how important it is that we give the antitoxin as early as possible.

From a clinical standpoint, diphtheria affects three organs principally: the kidneys, the heart, and the nervous system. The toxin probably affects the entire organism, but the organs mentioned are the ones that show the effects of the disease.

The first of these organs to be attacked are the kidneys. During the first week, we will find, upon the examination of the urine, albumin and casts, sometimes in large quantities. Cushing tells us that this is a degenerative nephritis found in all cases, and in serious cases causing a diminution in the amount of urine passed, with albumin and casts, but never causing edema or uremic symptoms. This form of nephritis does not produce marked symptoms and clears up within a few weeks, leaving no permanent effects.

The next organ to be affected is the heart. The toxin attacks the heart almost as soon it does the kidneys. The heart complication is far more serious than that of the kidneys. The most common cause of death from diphtheria is heart failure. Cardiac failure following diphtheria has for a long time attracted considerable interest as to its cause, but it is only within the last few years that we have proved that the failure is due to degenerative myocarditis. The occurrence of this myocarditis is apparently as constant as the nephritis, but is harder to demonstrate. Only through changes in color of the skin and the character of the pulse in mild cases does one suspect any trouble. Neither blood pressure nor cardiograph readings help any until the case is well advanced. Cardiac dilatation, with vomiting, heart block, or sudden death, terminate the picture in the worst cases. The symptoms of myocarditis show themselves first on the fifth to the seventh day, and usually culminate on the tenth to the fourteenth day, although it is several weeks before the heart returns to normal. After the twenty-first day has passed, one should no longer fear cardiac breakdown, according to Cushing. Cushing also states that he has been able to follow up a number of the severe cases and that no case after one year showed any pathology of the heart.

Naturally, we are interested in the treatment of these cases, so I shall give you the treatment followed at the Alexandra Hospital in Montreal, Canada. In this hospital they give absolute physical rest, prohibiting as far as possible the movement of the limbs. If vomiting begins, all nourishment must be stopped for two or three days. Morphine is given if there is restlessness, but this rarely occurs unless the case is hopeless. Digitalis does harm by causing vomiting and increasing the tendency to heart-block. Other heart stimulants seem of little use. If the little patient can be tided over a few days, the natural processes of repair will effect a cure.

The third clinical manifestation of the toxin is upon the nervous system, and usually makes its appearance in the general paralysis occurring about two weeks later. Walshe was the first to demonstrate that these paralyses followed any definite form. He has shown us how the first effect of the toxin on the nervous system is local, causing a paralysis at the site of the lesion, then passing along the course of the nerves to the central nervous system, affecting first the local nerves, then the neighboring nuclei, and then the general nervous system as a whole. The paralysis almost invariably culminates at the end of the sixth week. The treatment of this condition is principally rest and nutritious diet. The prognosis as regards life depends largely upon the involvement of the diaphragm and the muscles of respiration. Fortunately, these muscles are the last involved and their paralysis is short lived. If one can keep the patient alive for a week, all symptoms will subside, for in this, as in all other toxic sequelae of diphtheria, recovery is absolute and complete in the process of time.

If this clinical picture be a true one, it will be of benefit to us in managing our diphtheria cases, first in beginning treatment early with sufficient antitoxin to check the disease and neutralize the toxin in the circulation; second, by testing the urine during the first week to determine the amount of toxins absorbed; third, by watching the heart during the first and second weeks; fourth and finally, during the fifth week, in guiding the patient over the dangers of respiratory paralysis.

DISCUSSION

DR. M. K. WYLDER (Albuquerque, N. M.): I think we owe a vote of thanks to Dr. Milligan for working up this question. It may seem to a great many of you that we ought to know all about diphtheria, and I must say it is a shame to note the death rate on this account, when we know how to prevent this disease and that, if one has recently been exposed, a small shot of antitoxin will develop permanent immunity. No child should ever die because of diphtheria. Just as a baseball player muffs a fly ball which comes out in his field

and makes an error, so is every death from diphtheria due to some error. Either the doctor or the nurse has erred. The nurse sometimes thinks the condition is only a sore throat and does not give the doctor a chance, but if, when the doctor arrives, he does not work quick enough, then it is his error and his name should be put on the blackboard. Dr. Milligan covered the field very thoroughly and beautifully. In regard to the antitoxin, many doctors feel that they are not able to start with it until they have a positive diagnosis; and they do not realize the importance of giving it early. They say, "We will take a culture and when we get the report, if positive, will give a dose of antitoxin". By this delay, they may be throwing away the opportunity to save the life of the patient. It is not essential to await the culture; give the antitoxin. It is far better to give it and not need it than to need it and not give it. There is no better treatment for the septic sore throat than a big dose of antitoxin, anyway. With the ordinary cases, give 10,000 units intravenously and, if given early, you will not have the complications Dr. Milligan described.

DR. A. S. LATHROP (Santa Fe, N. M.): Dr. Milligan's paper was so excellent and complete that I do not see where I can add anything of interest. One thing, however, has not been stressed and that is the importance of taking a Schick test on children anywhere from three to six months after the antitoxin has been given. Figures show that only about 75 per cent are given immunity; the other 25 per cent are susceptible to diphtheria. Dr. Wylder spoke of the importance of giving antitoxin early—that cannot be emphasized enough. Ten thousand units the first or second day will have much more effect than fifty thousand on the fourth or fifth day of the disease. Complications are avoided by giving it early. Another point is that, in cases we do not see until the fourth or fifth day, the importance of rest cannot be overemphasized. Those whose body tissues have already been poisoned by the toxins need considerably more rest. Diphtheria is much more serious in rural areas than in cities, as in cities they build up their own immunity sooner than in the country.

DR. J. R. EARP (Santa Fe, N. M.): I merely want to mention that we publish helpful articles on public health questions every week—about sixteen English papers and all the Spanish papers are printing, regularly, rules to be observed and the importance of having children immunized. If any of you know that your local newspapers are not receiving these items, if you will let us know, we will gladly furnish them to every editor who can be persuaded to accept and publish them. In regard to reports from the Laboratory, just because a report may be negative do not get the assurance that your case is not diphtheria. There may be a slip somewhere between the taking of the swab and the Laboratory. They may not survive the journey. I know of one or two cases of death from clinical diphtheria, in which the Laboratory sent out negative reports. If you have cases of diphtheria in which you get negative reports from the Laboratory, I wish you would let us know. We want to go over the reports and want them to be accurate. Finally, I should like to say a good word for toxoid. I believe this is going to be very much better and give better results, as you can make your test first and adapt your treatment accordingly. We want to protect the younger children, but what is the right time to give them this immunization? After all, it is the one to three to five-year-old children who are dying from diphtheria. Therefore, if we start with the first birthday, we are not going to lose so many children. In New York, the State Health Commissioner sends out instructions to every parent nine months after a child is born. Maybe that would catch practically all our deaths from diphtheria in New Mexico.

DR. C. A. MILLER (Las Cruces): I had quite an experience lately—had seven cases of diphtheria in one family. There was only enough antitoxin for four of the cases and, when a further supply was received, two of the cases were so much better, I decided to wait, and the third case had been in progress so long I questioned the advisability of giving it, so did not give it to these three. It was difficult to decide what to do, yet those three got well. In another case I failed to send in a swab on a young man, and did not think it was diphtheria, but nevertheless gave antitoxin. He apparently got no results from the antitoxin, contrary to what has been said about antitoxin usually affecting a throat case, yet afterwards he was proved to be a carrier, as another case developed, according to the County Health Officer, which had been in contact with this one, and died.

DR. G. C. LINGENFELTER (Denver): I was very much pleased with Dr. Milligan's paper and want to emphasize all that Dr. Wylder said about not being afraid to give antitoxin too soon, or in too large a dose. You are not going to overdo it, but you may under do it. The remarks about smears are quite true—we do not always get accurate reports, and it is a wise method to be not quite satisfied if you have a negative report. There may have been some interference by the family in the way of having the child gargle, or swabbing the throat out with an antiseptic, shortly before the culture was taken and, hence, a negative report. Do not be satisfied with one negative. Take cultures over again and take them from the nose as well as from the throat.

DR. MILLIGAN (closing): I am very glad to have had such a generous discussion of this subject. Dr. Lathrop brought out the necessity of the Schick test on children in from three to six months after antitoxin has been given. That is a fine thing to do and should be done in all cases, but it is not practicable out in the rural districts. You have a great deal of difficulty to get these people to come in for their immunization, as transporta-

tion facilities are not good. They may live forty or fifty miles away and, when they come to the doctor's office, they do not like to have the office girl or the nurse take charge—they want the doctor. The public health nurses are always willing to cooperate with us. If we had more full-time health officers and nurses, we could arrange these patients in groups and meet them in the schools. In regard to the case that was rather virulent, where the doctor gave antitoxin without result, perhaps this patient was very susceptible to diphtheria and it worked faster than the antitoxin, so that the patient would require a much larger dose than is ordinarily given. As far as swabs are concerned, I am not in a position to say. I have received both positive and negative reports, and it has been my practice, when a case is suspicious, to go ahead and give antitoxin without paying much attention to the result of the culture.

BRAIN ABSCESS AND MENINGITIS FROM TRAUMA
(Fractured Skull)
Case Report

ERNEST B. THOMPSON, M. D.
El Paso, Texas.

(Presented at Staff Meeting of Masonic Hospital May 13, 1932.)

The patient, male, age 15, was injured March 6, 1932, when he collided with an automobile while riding his motorcycle. Upon being brought to the hospital, unconscious, he was seen to be regaining consciousness and suffering from the following injuries: a split wound of the right temporal region about three inches long, with a depressed fracture of the temporal bone extending into and involving the orbit. Brain tissue was observed to be exuding from this wound which was very ragged and dirty. On the right knee there was a lacerated wound about eight inches long over the patella, extending down to the patella ligament, which was partially torn from its tibial attachment.

First aid was rendered. The wound in the head was cleansed of debris, drains were inserted and lightly sutured. The knee wound was cleansed, also, and sutured. Tetanus antitoxin was administered. As a considerable degree of surgical shock was present from the accident, nothing further was done, and he was put to bed, made warm, and given stimulants.

He reacted well and immediately began a most satisfactory convalescence. On the day following the accident his condition was markedly improved. Examination of the right eye by opthalmologist disclosed that there was fixation of the globe from the hemorrhage into the orbit, with hyperemia of the disk. The prognosis regarding vision in this eye was precarious.

The general condition of the patient improved daily and on April 2 he was up in a wheel chair, with no complaint, and anticipating going home in a few days. The eye had cleared up nicely and promised normal function, subsequently. The wound in the knee was completely healed, as was the head injury. Check-up x-rays showed the depressed fragments of the temporal bone, as at first, but as no symptoms from this had manifested themselves, and as he was convalescing so satisfactorily, it had not been deemed wise to instigate surgical procedure on the skull.

On the morning of April 2, as stated above, he was feeling good and without complaint. Suddenly an excrutiating headache seized him and vomiting appeared, with rising temperature. He was observed to be rolling restlessly in bed, with his face an ashen color, and his neck was seen to be very rigid. Immediately lumbar puncture was done, and the spinal fluid withdrawn was turbid and yellow, under considerable pressure. It was seen to contain much pus.

With the impression of brain abscess and meningitis from extension, immediate operation was decided upon and this was done under local anesthesia. The old wound was opened and, upon incising the dura, a large amount of pus welled forth. Several pieces of bone were removed, also. Drains were inserted into the abscess cavity beneath the dura and the wound was closed.

Prompt improvement was noticed following this operative procedure. The headache was relieved and the patient's general condition was better. On the following day, his spinal fluid was clear and under no pressure. The stiffness of the neck was much less marked.

Then, after one day of improved condition, all of the former unfavorable symptoms again manifested themselves and the patient was seen to be in a grave state, growing worse all the while. And despite frequent injections of glucose intravenously, hypodermoclyses, infusions of glucose and saline intravenously, and transfusions of whole blood, his coma slowly deepened and death ensued on March 6.

Cultures from the first two withdrawals of spinal fluid were negative, even though they contained much pus and were under pressure. Culture of the third withdrawal was positive for Staphylococcus aureus. At time of operation cultures made from the pus from the brain abscess and from the bone sequestra were both positive for this same organism. Following the operation cultures of the spinal fluid were again positive for Staphylococcus aureus.

This case is interesting in that it shows no apparent damage from the loss of considerable brain tissue at time of accident, and the rapidity and suddenness with which the symptoms of a large brain abscess, with meningeal extension, may appear during the convalescence from the accident, one month later.

CASE REPORT

FELIX P. MILLER, M. D.
El Paso, Texas.

(Presented before the Staff Meeting of the Masonic Hospital, June 9, 1932.)

Patient, female, 10 years old, was brought to the hospital in a severe state of shock, with history of fall from a horse two hours previously. Patient was pale, skin cold and clammy. Abdomen was rigid and muscles hard. Patient complained of no nausea or vomiting. A diagnosis of injury to the abdominal viscera was made. Anterior, posterior, and lateral x-ray pictures were made of the spine, ribs and abdomen. No air bubbles were seen under the diaphragm. No injury to the ribs or spine was found. The left side of the pelvic bone apparently had been moved inward, outline was not symmetrical compared with the right, but no fracture or dislocation of the pubic bones was seen. A small amount of blood was noticed on the vulva, but there was no free bleeding from the urethra.

A small catheter was inserted into the urethra and a large amount of blood and urine obtained. The catheter was left in place. The abdomen was opened by a midline incision, extending from the umbilicus and going down over the pubic bone. After the fascia was incised, a hematoma was noticed at the lower angle of the wound. This portion of the abdominal wall was retracted and a large hematoma was found in the Retzius' space, extending from the reflex of the peritoneum well down under the pubic arch. This was removed by suction. Bleeding from the veins in this space was controlled by small gauze sponges, extending low in the wound. A rupture was found, extending along the anterior urethra and about an inch above the neck of the bladder. The catheter could be seen in the urethra. It was pulled well into the bladder wound. The urethra was closed over the catheter by means of No. 1 plain catgut, sutures going through all coats. The upper sutures were left long and used for retraction purposes in order to bring the lower wound into the urethra from beneath the pubic arch. The closure was continued downward to the end of the laceration in the urethra. The wound in the bladder was partially closed with chromic catgut with sutures and did not penetrate the bladder mucosa. This line of suture was enfolded and covered with No. 1 chromic catgut, placed after the fashion of Lambert sutures to enforce the first suture line. Mattress sutures were placed on each side, carried forward to attach the urethra to the tissues beneath the pubic arch. A purse string suture was passed in the upper end of the bladder wound. A suprapubic drain was fastened into the bladder. This tube was held firmly. A second purse-string suture, which penetrated the tube to fix it in place, was passed. Two penrose drains were inserted on either side of the urethra well down in the Retzius' space. Fascia was approximated with No. 2 chromic catgut, reinforced with silkworm sutures. Urethral catheter was sutured to the labia majora to prevent its being dislodged.

It was felt that the abdomen should be explored so a high right rectus incision was made. Free blood was found in the peritoneal cavity. There was a laceration through the peritoneal coat of the bladder about 3½ inches long. The muscular coats were not lacerated. All the abdominal organs were examined, no injury found. The blood was removed by suction and the wound was closed without drains. Patient was given saline by hypodermoclysis while on the operating room table.

Both the suprapubic and the catheter drains functioned nicely.

Patient reacted with a slight rise in temperature, first few postoperative days. Has been able to take fluids. Has had some discomfort from gas. There were no untoward symptoms.

The penrose drains were removed on the fifth postoperative day, and the suprapubic drain on the eleventh. The catheter was allowed to remain and functioned normally. The suprapubic wound closed by granulation with slight urinary drainage for several days.

CASE REPORT

R. J. STROUD, M. D.
Tempe, Ariz.

(Presented before the Staff Meeting of the Good Samaritan Hospital, Phoenix, April 25, 1932.)

Case No. 13549: Male, aged 42, married, presented himself, January 12th of this year, complaining of feeling tired and saying that he had not felt well since early fall. He had not been able to stand hard work of any kind, and had some indefinite pains all over the body.

Examination revealed a man who had lost some weight, who was still in fairly good condition and well nourished. There was some pallor, but he said that it was more or less a normal condition. Outside of a somewhat tender area at the right kidney, nothing of importance was found. A urine test showed no abnormalities, and pus was looked for at the time.

The main thing in the patient's history was the fact of a double pneumonia 12 years ago, followed by an abscess of the right lung and spontaneous drainage through the bronchial tract. He was sick a long time but finally recovered. He has had no diseases, since, that relate even remotely to this present condition.

He did not report back, and, on Feb. 6th, I was called at night, his wife saying that she thought he had appendicitis. Examination revealed temperature of 102.6° and pulse of 80. The pain was rather higher than the appendix and came downwards from the right kidney region. Murphy sign was positive on the right side. The pain was not transmitted to the inner side of thigh, penis, or

testicle. The most tender spot was just under the twelfth rib. The urine showed a trace of albumin and was loaded with pus. Some blood cells were also present.

The course of the disease was rather stormy and a diagnosis of pyelitis was made. For the first four days, patient's temperature ranged from 102° to 104°, the evening temperatures always being higher. The pulse rate was between 76 and 92. He did a good deal of belching, but there was no vomiting. He was given amytal for pain and at times an opiate was necessary. After a week the pus in the urine had dropped very materially and his temperature began to come down until the 19th, when it was normal all day.

All of the time the pain in the right back was present, but left, except for a slightly uncomfortable feeling, the 18th and 19th; but the pulse had remained in the 90's, while his temperature dropped. The family was told that pus was probably outside the kidney and that an operation might have to be done. Heat, urinary antiseptics, water and alkalis were taken. Nonspecific proteins were given intramuscularly and infrared heat prescribed. On the 20th there was a slight trace of albumin in the urine, with a dark heavy output, slightly scanty, but no pus. The morning temperatures were normal, with an evening rise to 101°, until the morning of the 23rd. The patient looked and acted better, although during all of this time he was toxic enough to be more or less irrational, which could not be attributed to the amytal or occasional opiate. At the same time he would answer questions correctly and promptly, but the family described his symptoms as talking at random when not engaged in definite conversation. On the morning of the 23rd he had a sudden chill, temperature came up to 102.8°, with high pulse, and he looked very sick and toxic.. He was immediately sent to the hospital with a diagnosis of perinephritic abscess, pyelitis, and some bronchopneumonia. He was x-rayed that day for abscess around the kidney or possible kidney stones. The report was negative although, in the light of subsequent events, the gas bubble described could have been the outline of the abscess.

That night the patient suddenly became dyspneic, with shallow breathing, and was cold and in a clammy sweat, with inperceptible pulse. He looked as if he were going to die. I was on my way to see him when the night supervisor tried to call me about this sudden collapse. He was given 1700 cc. of saline solution reinforced with 3½ drachms of tr. digitalis and 60 grams of glucose. The pulse returned under my finger, while this was being given, to 120. His breath was stercoraceous at the beginning but cleared up by the next day.

The lungs showed signs rather hard to determine, being due to upward pressure of the pus, so pictures were ordered on the 25th with reference to the lungs, and showed high diaphragm. By inference this was due to pus pressure from below. An operation was advised, but before operating I asked for consultation. Dr. Greer was called and advised a preliminary needling. When this was done, some 16 to 18 ounces of pus were withdrawn and the patient felt a great relief. His condition being as it was, the operation, done under novocaine, was that of following the needle, which had been left in position after drainage in his room, and going upward and inward with blunt instrument, spreading the tissues until the cavity was reached and a drain inserted. There was no pain or shock to the procedure and his condition improved rapidly. He was rational in all ways the next day, and said that he felt like himself more than at any time since being sick. From the 26th until March 3rd, he did well and, while thin and yellow from the prolonged toxemia was feeding himself and asking when he was to return home. On the 4th the drain tube was reinserted in the back, the old one having come out while he was being dressed. Early in the morning of the 5th, he had a sudden chill, with a rise of temperature to 103.8°, and the pulse and respiration took on the characteristic curve of a lobar pneumonia. Signs in the right lung were not much greater than when the abscess was present, but an x-ray was done to see if there were lung changes not found by stethoscope. X-ray revealed a mass in the hilus extending to the lower part of the lung. There is still some indefiniteness as to what it was. Dr. Watkins and I went over the thing and, as there was a possibility of fluid encapsulated or pus, needling was done and none found. One of the fine needles was broken off in the attempt, and is still there. Dr. Mills thinks it is between the pleura and ribs. Crosscutting following x-ray did not reveal it on the upper surface of the rib. This needle was a fine novocaine one, and its tendency to bend when meeting an obstruction has told me it is a poor thing to use for this procedure. On the 9th the temperature dropped very suddenly late in the day and next morning was subnormal when the patient had another collapse and again responded to saline solution intravenously. On the 11th a transfusion was done. It did not seem so necessary at this time, as he had regained a lot of strength, but it picked him up a bit more. On the 10th saline and glucose had given him a sharp reaction, so it was thought inadvisable to submit him to another possible reaction from transfusion. I will say that he was typed the first day he entered the hospital and a donor was found. A big reaction followed the transfusion. When the chill and rise of temperature began on the 5th, he was put on supportive treatment and inhalations as for pneumonia, immediately. As he had been getting some digitalis before, the heart rate soon dropped low and stayed there with a daily dose. When he had the cold clammy sweat of the crisis he was first wrapped up with hot water bottles. However, the whole back was exposed to infra-red for many hours, which dried him up, made him feel more comfortable, and brought his temperature up to normal. I think it beats hot water bottles and blankets. His

recovery was rather slow and he has not yet returned to work. There is still a discharging sinus at the site of operation.

This case illustrates the need of consultation between the clinician and the roentgenologist when a picture is taken, with a full description to the x-ray man of what he may expect to find if the clinical signs denote pathology in any particular place. We should have saved this man two days of profound toxemia on the first picture if we had been more closely associated on the thing.

I am having smears made of the pus from the sinus, and also cultures, at the present time.

Without the history of a blow or any other focus, is it possible that the old lung abscess still carried some organisms that found their way to the kidney region?

THE NEW MEXICO MEDICAL SOCIETY

(Continued from September Issue)

Afternoon Scientific Session

Dr. Earl D. McBride, (Oklahoma City, Okla.) opened the afternoon session with a paper on "Arthritis—Under the Light of Recent Research." Dr. McBride advised that the classification adopted by the American Committee for the Control of Rheumatism be studied and adopted by the profession; that the viewpoint of the clinician should be broad and his treatment thorough; that the sensitivity theory is rational and serologic therapy must be directed with an appreciation of allergic phenomena; and that the specific vaccine selected by means of the complement fixation text is reliable and deserves more general trial.

In discussion, Dr. R. W. Mendelson (Albuquerque, N. M.) agreed with Dr. McBride that each case is a personal equation and should be considered individually, and believed that the day of intravenous medication has passed.

Dr. V. E. Berchtold (Santa Fe, N. M.) stated that he was more or less interested in arthritis because he had suffered from it several times himself and he knew that specific vaccine in small doses adds a great deal to the comfort of the patient, compared with the intravenous injection of vaccines.

In closing, Dr. McBride explained results obtained from the vaccine treatment as dependent upon the attitude of the patient; that sometimes it is very slow and may take from three to six months to a year before improvement is shown. so the physician should endeavor to convince his patient that he will not and need not expect spectacular relief, but rather a slow and tedious process. If faithfully continued from six months to a year, tissue resistance will gradually be built up against the specific organism, and results will surely follow.

Dr. Hugh Jones (Los Angeles, Cal.) delivered an extemporaneous address, in connection with lantern slides, showing various types of bone tumors, stressing particularly osteochondroma.

In discussion, Dr. C. W. Irish (Pasadena, Cal.) recalled the case of a girl of 16, who suffered a psychic trauma and developed psychotic symptoms very similar to those of dementia precox. A cystic tumor was finally taken care of and the psychotic symptoms cleared up.

Dr. William C. Menninger (Topeka, Kansas), in a paper on "The Neurotic Patient: The Ever Present Problem", summarized thus: "In the neuroses we are dealing with a very wide field of medicine for the reason that all of us have neurotic tendencies. When the pressure of the tendencies becomes too great, we have to compromise with these tendencies and we may develop a neurosis. We regard it as always a compromise between the neurotic tendencies and the situation, which is acceptable, but entirely unconscious in its workings, to the individual. These unconscious conflicts express themselves in bizarre ways, almost always with pain or distress but, because they find expression, which is fundamentally their aim, the individual is in a sense satisfied. His unconscious is satisfied and it does not care to make any change. So, despite the strivings of the conscious to get well, the more powerful unconscious does not desire to get well. In the final analysis, recovery is possible only when the intrapsychic conflicts are resolved."

Discussion was opened by Dr. A. B. Stewart (Las Vegas, N. M.), who commented upon statements made earlier in the day by cancer experts, that over 120,000 persons die eachy ear from certain types of cancer alone, and referred to the fact that neurologists and psychiatrists state there are over 75,000 commitments annually to public institutions for the control of the insane. "There are born each day in the United States 7,000 infants, of which, under present statistics 270 will spend their last days in public institutions for the insane." This shows us our tremendous responsibility toward the class of individual we are more inclined to push away.

Dr. C. W. Irish (Pasadena, Cal.), complimented Dr. Menninger upon his general summary of the modern up-to-date treatment of neuroses, stating that the majority of medical men like to escape from the neurotic patient, who takes up more time and patience than any other type. He wondered if the profession and public realize the immense economic loss suffered in each community by the mental complex and estimated that it could bring about as much economic loss as the patient with a very decided organic lesion.

Dr. J. R. Earp (Santa Fe, N. M.) expressed the belief that it might be possible for the troublesome neurotic patient to become an entertaining subject to the busy doctor and, instead of being a nuisance, become an interesting hobby. He recommended a book by Hart, "The Psychology of Insanity", as very valuable in giving excellent ideas on the subject and telling how to handle this tpye. He sug-

gested that thought be given to the mental hygiene movement and the prevention of the development of neuroses, as it might be possible to prevent a good deal more successfully than cure, if more attention is paid to the beginning of neuroses while in the mental hygiene stage.

Dr. H. T. Jones (Los Angeles, Cal.) referred to the slight slur cast upon the general practitioners for the way in which they receive this class of patients and stated that at school most men had been taught never to diagnose from history, until everything else had been investigated. He asked what could be done if a woman comes into the office, throws her arms around the doctor's neck and commences to cry. If the doctor stops to listen, he is apt to be severely criticized, and, on the other hand, is also criticized if cruelty is necessary and is practiced.

In closing, Dr. Menninger expressed the belief that what to do with a lady when she throws her arms around your neck seemed entirely dependent upon the individual, but he felt it always safer to have a desk between himself and the patient—then, unless they have long arms, one is safe.

Dr. Curt von Wedel (Oklahoma City, Okla.), the next speaker, presented a very interesting paper, and demonstrated with lantern slides different phases of work in connection with "Plastic Surgery", touching principally upon that phase which has to do with the transplantation and shifting of skin. The Doctor asserted that "there is no sure way to success, but if fundamental rules are followed, skin transplantation today is highly practical. With the exception of the transplantation of very large full-thickness grafts on uneven surfaces, we today can almost promise success. Our failures are very few and far between and even with a transplant of a rather large area of full-thickness free graft, we seldom lose all the graft and are quite successful in the majority of cases."

In discussion, Dr. L. E. Griswold (Oklahoma City, Okla.) said in behalf of Dr. von Wedel's work that the profession in Oklahoma City were very proud of his success; that he has a number of cases all the time and his results are always excellent.

Dr. W. H. Woolston (Albuquerque) expressed his appreciation of Dr. von Wedel's interesting and instructive paper and commented that the work, which is extremely tedious, required much care and patience.

"Immunization and Treatment of Diphtheria" was the subject of the next speaker, Dr. C. F. Milligan (Clayton, N. M.), who asserted that "Diphtheria begins as a localized disease, usually in the throat, and later becomes more general in its action. The toxin is a slow poison, taking some weeks to produce all of its effects. From a clinical standpoint, diphtheria affects three organs principally, the kidneys, the heart, and the nervous system. The toxin probabily affects the entire organism but the organs mentioned are the ones that show the effects of the disease. The treatment followed at the Alexandra Hospital, Montreal, Canada, is absolute physical rest, prohibiting as far as possible the movement of the limbs. If vomiting begins, all nourishment must be stopped for two or three days. Morphine is given if there is restlessness, but this rarely occurs unless the case is hopeless. Digitalis does harm by causing vomiting and increasing the tendency to heart-block. Other heart stimulants seem of little use. If the little patient can be tided over a few days, the natural processes of repair will effect a cure. The third clinical manifestation of the toxin is upon the nervous system, and usually makes its appearance in the general paralysis occurring about two weeks later. Walshe was the first to demonstrate that these parlayses followed any definite form. He has shown us how the first effect of the toxin on the nervous system was local, causing a paraylsis at the site of the lesion, then passing along the course of the nerves to the central nervous system, affecting first the local nerves, then the neighboring nuclei and then the general nervous system as a whole. The paralysis almost invariably culminates at the end of the sixth week. The treatment of this condition is largely rest and nutritious diet. The prognosis as regards life depends largely upon the invlovement of the diaphragm and the muscles of respiration. Fortunately, these muscles are the last involved and their paralysis is short lived. If one can keep the patient alive for a week, all symptoms will subside, for in this, as in all other toxic sequelae of diphtheria, recovery is absolute and complete in the process of time. If this clinical picture be a true one, it will be of benefit to us in managing our diphtheria cases: first, in beginning treatment early with sufficient antitoxin in the circulation; second, by testing the urine during the first week to determine the amount of toxins absorbed; third, by watching the heart during the first and second weeks; fourth, and finally, during the fifth week, in guiding the patient over the dangers of respiratory paralysis."

Discussion was opened by Dr. M. K. Wylder, (Albuquerque), who commented on the death rate due to this disease as a shame, when we know how to prevent diphtheria and that, if one has recently been exposed, a small shot of antitoxin will develop permanent immunity. In regard to antitoxin, many doctors feel they are not able to start with it until a postive diagnosis has been made, and they do not realize the importance of giving it early. It is not essential to await a culture—give antitoxin. It is far better to give it and not need it than to need it and not give it. With ordinary cases, give 10,000 units intravenously and, if given early, one will not have the complications Dr. Milligan described.

Dr. A. S. Lathrop (Santa Fe) stressed the importance of the Schick test on children from three to six months after antitoxin has been given, as figures show that only about 75 per cent are given immunity, the other 25 per cent being susceptible

to diphtheria. "In cases we do not see until the fourth or fifth day, the importance of rest cannot be overemphasized, as those whose body tissues have already been poisoned by the toxins need considerably more rest than others."

Dr. J. R. Earp (Santa Fe) called attention to the fact that the New Mexico Bureau of Health has published helpful articles on public health questions weekly in sixteen English papers and all the Spanish papers, and is constantly stressing the importance of having children immunized. These items will be gladly furnished to any editor who can be persuaded to accept and publish them and, if your local newspapers desire them, please advise the Bureau. Dr. Earp also cautioned that, just because report from the laboratory on a culture might be negative, it should not be considered conclusive that the case was not one of diphtheria. He urged protection of the younger children and thought immunization, if started about the time of the first birthday, would prevent many deaths, as most of these occurred in children from one to three years old.

Dr. C. A. Miller (Las Cruces) recited an experience he had recently—seven cases in one family. There was only enough antitoxin on hand for treatment of four of the cases and, when a further supply was received, two of them were so much improved that he decided to wait and the third case had been in progress so long that he questioned the advisability of administration, so did not give it to the three, and all recovered. In another instance where he failed to send in a culture, not thinking the case one of diphtheria, the patient apparently showed no results from antitoxin which was given, yet was afterward proved to be a carrier.

Dr. G. C. Lingenfelter (Denver) emphasized Dr. Wylder's remarks in not being afraid to give antitoxin too soon, or in too large a dose, stating that there is more danger of underdoing than overdoing in this regard. He also advised against being satisfied that a case is negative simply because the laboratory report might be negative, suggesting that cultures be taken over again and from the nose as well as the throat.

In closing discussion, Dr. Milligan expressed his appreciation of same and agreed that it would be very advantageous if a Schick test could be made on children from three to six months after antitoxin had been given, but thought it not practicable in the rural districts, where it is very difficult to get the people to come in for their immunization, much less afterward, as transportation facilities are not good. They may live 40 or 50 miles away, so it is advantageous, if possible, to set some afternoon and send word far enough in advance to have them all come in at that time. Otherwise, if they come in at various times and do not catch the doctor in his office, they do not like the office girl or nurse to give the inoculations. If we had more full-time health officers and nurses, we could arrange these patients in groups and handle them without difficulty. In regard to the case that was rather virulent, where the doctor gave antitoxin without result, perhaps this patient was very susceptible to diphtheria and the toxin worked faster than the antitoxin, so that larger doses than those ordinarily given would be required.

Adjournment until 9:30 a. m., May 21, 1932.

Meeting of House of Delegates

A meeting of the House of Delegates was called at the close of the Scientific Session, there being present:

President, Dr. F. D. Vickers (Deming); Secre-

(Continued on page 434)

PUBLIC HEALTH NOTES

J. ROSSLYN EARP, DR. P. H.
Director New Mexico State Bureau of Public Health.

RELAPSING FEVER

We were honored recently by a visit from Dr. Emile Brumpt, professor of parasitology in the University of Paris and author of several well-known text books, including "Les Mycetomes," "Precis de parasitologie," "Spirochetoses," "Trypanosomes humaines, bilharzioses" and "Travaux pratiques de parasitologie."

Dr. Brumpt had been searching in the dust of New Mexico for the tick Ornithodoros which carries relapsing fever. He had not found a specimen, which does not, of course, prove that there are no specimens to be found. Relapsing fever (African tick fever) is common in old Mexico, has been found in Arizona[1], Colorado[2] and California.[3] It was first traced to the tick Ornithodoros turicata in this country by Malcolm Graham in Texas.[3] It has recently been reported in Nevada.[4] Dr. Brumpt tells me that the chipmunk has been found to carry the infection in California, so confirming Graham's prediction, made in 1931, that there must be an animal reservoir of the disease. With recognized cases of relapsing fever on all our frontiers, we shall be exceedingly lucky if we escape without any cases in New Mexico. For this reason, relapsing fever will have been added to the list of notifiable diseases in this state by the time this article appears in print.

Psittacosis will be made a notifiable disease at the same time.

WHOOPING COUGH

Last year was a year of exceptional diphtheria incidence in New Mexico. Even so, there were fewer deaths from diphtheria than from whooping cough, which serves to emphasize the great importance of protecting infants from infection with B. pertussis.

Table I shows plainly that the first four years of life are those in which we are most to fear this disease. Thirty-one out of forty-seven deaths occurred in the first year of life. Twenty-two of these infants were less than six months old, a finding which does not support the theory of immunity during the earliest months. The fact that reported morbidity is lower than mortality in this age group might be an expression of the great virulence of the disease in this age group but is more probably an indication of incomplete reporting.

Table I

Ages	Cases	Deaths
Under 1 yr.	26	31
1 "	32	11
2	23	2
3	25	1
4	34	2
5- 9	93	
10-14	15	
15-19	3	
20-24	1	
25-64	0	
65-69	1	
Age Unknown	15	
Total	268	47

Whooping Cough Morbidity and Mortality by Age Groups—New Mexico, 1931.

Fatality rates for the three most important contagious diseases are shown in Table II. When the fatality rates for New Mexico are compared with the following table of fatality rates given by Rosenau, one cannot avoid a suspicion that the number of cases notified is far short of the number occurring. This must not be taken as a reflection on the profession in New Mexico. There are many people in this state who do not consult a physician as often as they should.

Table II

	Cases	Deaths	Reported Cases per death	Fatality rate
Whooping Cough	268	47	5.7	17.5±1.56
Diphtheria	372	45	8.3	12.1±1.56
Measles	1379	34	40.6	2.5± .28

Fatality rates for Whooping Cough, Diphtheria and Measles—New Mexico, 1931

Table III

	Denmark	England	U.S.A.
Whooping Cough	1.4	1.3	0.7
Diphtheria	0.7	0.9	1.2
Measles	0.5	1.9	0.6

Fatality Rates (Rosenau)

WHOOPING COUGH VACCINES

Can we protect young children against infection? The history of whooping cough vaccines has been fraught with disappointment. Usually, when results have been statistically controlled, as in a recent report by Schowalter,[5] they have given no encouragement. Still individual physicians, as Ellen M. Kent Hughes,[6] remain convinced of its efficacy in their hands. There is, moreover, the controlled experiment of the epidemic in 1923 in the Faroe Islands, where the mortality among the vaccinated was only one-twelfth that among the non-vaccinated. Madsen, who obtains his vaccines from cultures grown on fresh blood medium, recommends three doses (subcutaneous or intramuscular), of 0.5 cc., 0.7 cc. and 1.0 cc., of a suspension containing 10,000,000 bacteria to the cubic centimeter.

Undoubtedly, ultimate success with this method depends upon the technic of preparation of the vaccine. Leslie and Gardner[7] killed their vaccine with 0.2 per cent formalin. Pierret[8] lays great stress on the doctrine that pertussis vaccine should not be killed by heat. He considers it important also that the vaccine should contain numerous strains of the organism and should be given in adequate dosage. He gives doses of 250 million bacteria, or even more, though he reduces his dose to one-half or one-quarter in children under three years of age.

While we are waiting for a perfected vaccine, it is of the first importance to isolate all well infants when whooping cough is about. Successful isolation of the well often depends upon successful diagnosis of the sick in the catarrhal stage, before the whoop develops. I find that some New Mexico physicians are unaware of the great aid they may have in diagnosis by getting the patient to cough upon suitable culture medium. The invisible droplets of an ordinary cough will develop into colonies of the Bordet-Gengou bacillus. Failure to find these colonies at the first attempt will occur in less than one-quarter of cases in the catarrhal stage. The state laboratory has assisted some physicians to make positive bacteriological diagnoses by supplying the medium.

NEW LABORATORY SERVICE

Miss Greenfield announces in the following paragraph an improvement in diphtheria diagnostic service which we hope will make less possible negative reports on cases that are clinically positive.

The laboratory is planning a new method of handling diphtheria cultures, hoping that we shall obtain more sensitive results in the control of diphtheria. We shall send out tubes of Loeffler's culture medium sealed with cellophane caps. The cellophane cap will prevent the medium drying too rapidly. Culture medium so preserved should be in a suitable condition for use even after several months' storage. This stock of medium must be watched carefully, however, and when it shows perceptible drying should be returned to the laboratory, when a fresh supply will be forwarded. Please return all tubes not used, and do not divert laboratory tubes to other uses. The laboratory is running on a very small budget, and cooperation in avoiding even small losses is earnestly requested.

Miss Greenfield is also experimenting with capillary tubes for the collection of serum from primary chancres, in order that diagnosis of syphilis may be made in the early sero-negative stage by direct dark-field examination. Physicians interested in this new service should write to Miss Greenfield at the New Mexico Pubilc Health Laboratroy, University of New Mexico, Albuquerque.

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BOOK REVIEWS

PULMONARY TUBERCULOSIS. By Maurice Fishberg, M. D., Chief of the Tuberculosis Service, Montefiore Hospital, New York City, and of its Country Sanatorium for Incipient Tuberculosis. Fourth edition of two octave volumes, 1191 pages illustrated with 232 engravings and 8 plates. Cloth, $15.00 net. Lea & Febiger, Philadelphia.

Volume I covers the etiology, pathogenesis, symptomatology, roentgenology and clinical forms. Volume II covers tuberculosis in children and the aged, tuberculosis of the pleura, diagnosis, complications and prognosis, medical, dietetic, climatic and institutional treatment, therapeutic pneumothorax, surgical treatment.

This standard work has been expanded, rewritten and revised to cover every recent advance in the subject. Over three hundred pages and over one hundred new illustrations, mostly roentgenograms, have been added. The text is clear, instructive and accurate. It presents the disease from every angle and is written in a style which compels attention and imports information. It is based on the latest and most authoritative knowledge. Every clinical aspect is stressed and roentgenology plays a much more important part than in previous editions. Every variation in the course of the disease is described and treatment, especially treatment in the home, is emphasized throughout, showing how it may be adapted to the individual case and conditions so as to make it more effective. This new fourth edition, like its predecessors, will be recognized as the most thorough and complete presentation of the subject.

Dr. Fishberg writes for both the general practitioner and the institutional worker. It is his belief that "incipient" does not invariably mean curable tuberculosis and, conversely, that "advanced" disease does not necessarily indicate a hopeless outlook. The work makes a sharp distinction between infection and disease and between tuberculosis and phthisis. It voices the conviction that sanatorium and climatic treatment are not the only methods of handling patients effectively and that careful home treatment under the direction of a qualified physician can produce immediate and ultimate results. The work reflects thirty consecutive years of experience with the tuberculosis problems of New York City.

Much of the material is entirely new, covering recent advances. There are new chapters on the medical phases of operative treatment, phrenicectomy, apicolysis, extrapleural thoracoplasty, oleothorax, etc. New sections are introduced covering the filtrable, ultramicroscopic and non-acid-fast types of tubercle bacilli, the etiological role of social and economic conditions, intraclavicular infiltrations, the differentiation and prognostic significance of apical as compared with subapical lesions, pulmonary cavities, tuberculous bacillemia, chronic and healed miliary tuberculosis, epituberculous infiltrations, the erythrocyte sedimentation test, the Wassermann test in the tunberculous, B.C.G. in prophylaxis, erythema nodosum, heliotherapy in pulmonary, intestinal and laryngeal tuberculosis, syphilis, asthma and hay fever in the tuberculous, the Gerson salt-free diet, insulin in tuberculous diabetics, chemotherapy, especially sanocrysin, the eugenic aspects of tuberculosis, etc. The exceedingly important work on the stages of tuberculosis initiated by the investigations of Ranke are considered in detail.

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THE SOUTHWESTERN MEETING

Albuquerque, December 8, 9 and 10

The program of the Eighteenth Annual Session of The Medical and Surgical Association of the Southwest, under the capable chairmanship of Dr. W. A. Gekler, of Albuquerque, is rapidly taking form. The other members of the Committee are Drs. David M. Davis of Phoenix, C. A. Thomas of Tucson, S. A. Schuster of El Paso, and M. K. Wylder of Albuquerque. The scientific program will be made up almost exclusively by members of the Association, thus coming back to the original purpose of the organization, which was to present, by papers and clinics, the scientific and medical work of the profession of the southwest.

The completed program will be published in the November issue of this journal, which will issue about November 15th. Among the papers already announced are the following:

Dr. W. P. Holbrook, Tucson, Ariz.: "Therapy in Chronic Rheumatic Diseases."

Dr. J. J. Beatty, Tucson, Ariz.: "What Can We Do with the Far Advanced Case of Tuberculosis?"

Dr. C. S. Kibler, Tucson, Ariz.: "Pulmonary Tuberculosis with Acute Onset."

Dr. Meade Clyne, Tucson, Ariz.: "Scalieotomy in Treatment of Tuberculosis."

Dr. M. C. Comer, Tucson, Ariz.: "Conservatism vs. So-called Radicalism."

Drs. John W. and Zedud M. Flinn, Albuquerque, N. M.: "The Tissue Reactions to the Tubercle Bacillus."

Dr. M. K. Wylder, Albuquerque, N. M.: "Autumn Diarrhea in Children."

Dr. H. M. Purcell, Phoenix, Ariz: "Trichomonas Infection."

Dr. W. Warner Watkins, Phoenix, Ariz.: "The Natural History of Gastric Ulcer as Shown by X-ray."

Dr. Louis B. Baldwin, Phoenix, Ariz.: "Some Clinical Aspects of Essential Hypertension."

Dr. Henry L. Franklin, Phoenix, Ariz.: "Refractive Errors in Their Relation to General Medicine."

Dr. J. H. Patterson, Phoenix, Ariz.: "The Cervix and Some of Its Problems."

Dr. Wm. L. Reid, Phoenix, Ariz.: "Symptoms of Gall Bladder Disease and Operative Findings."

Dr. W. P. Sherrill, Phoenix, Ariz.: "Tuberculosis from the Standpoint of the Pediatrician."

Dr. Palmer Dysart, Phoenix, Ariz.: "Presentation of a Transfusion Apparatus."

Dr. David M. Davis, Phoenix, Ariz.: "Radium Treatment of Carcinoma of the Prostate and Bladder."

Dr. Victor Randolph, Phoenix, Ariz.: "Intrapleural Pneumolysis, with Special Reference to Bilateral Pneumothorax in the Treatment of Pulmonary Tuberculosis."

Dr. Thomas W. Woodman, Phoenix: (Title to be announced).

Dr. J. H. Gambrell, El Paso: "Carcinoma of the Thyroid with Case Report."

Dr. Kelvin D. Lynch, El Paso: "Some Observations on Transurethral Prostatic Resection."

Dr. N. H. Keller, El Paso: "Physiology of Nephritis."

Drs. Stephen A. and Frank P. Schuster, El Paso: "Some Observations on Cataract."

Dr. R. B. Homan, Sr., El Paso: "Lung Abscess."

The hotel headquarters will be at the Franciscan. Other details of the program, such as clinics, entertainment and registration will be announced next month.

THE INITIATIVE ON THE ARIZONA INDUSTRIAL COMMISSION

The initiated measure to abolish the Arizona Industrial Commission is a matter which should concern the entire medical profession of that state. It is difficult to see how any impartially minded physician or surgeon in the state can support this measure. The policy of the Commission has always been one of cordiality and fairness to the medical practitioner. They made a poor start in the outset of their organization when, under the guidance of a legal advisor trained in California, a fee schedule similar to the one in that state was announced. However, when these fees were protested by the Arizona State Medical Association, they were invited by the Commission to write their own schedule. A special meeting of the Association was called, met in Phoenix, and worked out the present schedule; although this was more generous than can be found in any other state, it was adopted by the Commission. At the same time, the Association was requested by the Commission to appoint an advisory committee to confer with the Commission over any differences which might arise between that body and individual practitioners. Such a committee was appointed but failed to function when called on, so that the Commission was forced to work out its medical problems the best way it could.

The difficulties and frictions encountered in Arizona have not been any different from those found elsewhere, and they center about cases in which the Commission acts as insurer. Naturally, in that capacity, they have been directly interested in the type of service rendered injured workmen, and in the fees charged for those services. Where they have found the service poor and the charges high, the Commission has not hesitated to express itself, and this has created enemies in the ranks of the medical profession. Similar situations are found everywhere Industrial Commissions operate. They arise out of the more or less human desire to make insurance cases as remunerative as possible, even at the cost of the desideratum devoutly sought by the Commission, which is to return the injured workman to work as quickly as possible and with as little cost as possible. Where these two ideas clash, somebody is bound to be dissatisfied, but the remedy for this is not destruction of the whole machinery, but cooperation in its operation.

The disaffection in Arizona has been seized upon by individuals whose personal interest will be best served by abolishing the Industrial Commission, thereby making an "open season" of the injured workmen and their employees for the benefit of whoever can garner the most personal profit out of their misfortunes. Attempt is being made by these individuals to enlist the sympathy of members of the medical group in support of this initiative, although employees, employers, and the general medical profession of the state are opposed to such a destructive measure. Of course, there are occasional workmen who are not satisfied with the awards given, but, on the whole, the Commission has been not only fair but generous in these awards. If there are personal differences between individual members of the medical profession and the Commission, these should not be allowed to take precedence over the decision of the state medical organization that the Industrial Commission should be supported in its efforts to serve the workmen of the state, and this initiated measure should NOT be supported.

DR. R. J. STROUD, REPUBLICAN CHAIRMAN

Not content with furnishing the Democratic party its candidate for governor, Tempe, the little college town on the south bank of Salt River, has also contributed the Chairman of the Republican Central Committee of Arizona, in the person of Dr. R. J. Stroud. With an honorable record of service in the medical ranks of Arizona, Dr. Stroud is entering upon his new duties in the political arena with his customary ability. Among the services rendered the medical profession are included those as president of the Maricopa County Medical Society, national legislative committeeman of the State Association, as well as serving as secretary of the State Board of Health under Governor Phillips. It is interesting to note that our Republican presidents seem to have a preference for physicians in their cabinets. The Secretary of the Department of the Interior under President Coolidge and President Hoover has, in each instance, been a physician and an ex-president of the American Medical Association. This office was filled under President Coolidge by Dr. Hubert Work and the same place is now held by Dr. Ray Lyman Wilbur. Dr. Stroud's experience as an abdominal surgeon might qualify him for the same position. (Sic.)

Physicians of Arizona have been willing to give time to public service, whenever called upon, and the roster of those who have so served would be a long one. Dr. Stroud has given excellent service in several minor capacities and we know that he will bring to his new responsibility his characteristic vigor and good judgment.

PERCY GILLETTE CORNISH, SR.

(Albuquerque)

In the passing of Dr. P. G. Cornish, Sr., another of the noted pioneers of the profession in the Southwest passed from the scene of this world, leaving a host of friends in all walks to life, to join with his sorrowing family in their mourning.

His death occurred on September 28th in the Good Samaritan Hospital in Los Angeles, where he had undergone an operation. Dr. P. G. Cornish, Jr., arrived by plane from Albuquerque in time to be with his father several hours before the end. The body was brought to Albuquerque for last honors by the community and various organizations of which he was an active member.

Dr. Cornish was born in 1857, a native of Alabama. He received his earlier education in Kentucky and graduated in medicine from Jefferson Medical College in 1885. He moved to Arizona in the early nineties, being located for a time at Flagstaff. He married in Prescott during this period, his wife being from the Coffin family, still pioneers in Phoenix; she died in February of this year. Dr Cornish moved to Albuquerque in 1897, and thrroughout his long and honorable career in New Mexico endeared himself to the profession of the Southwest and to the citizens generally. For years he was chief surgeon for the Santa Fe Railroad in New Mexico. He has served as president of the New Mexico Medical Society and of the Medical and Surgical Association of the Southwest; he was a Fellow of the American College of Surgeons and of the American Medical Association. He was a charter member of the Albuquerque Chamber of Commerce, has been eminent commander of the Knights Templar, member of the Shrine, of the Elks, and a faithful member of the Presbyterian Church, the Rotary Club and other organizations. He is survived by his son, Dr. P. G. Cornish, Jr., of Albuquerque, one brother who resides on the family homestead in Alabama, and the members of the family of his wife.

THE NEW MEXICO MEDICAL SOCIETY

(Continued from page 429)

tary-Treasurer, Dr. L. B. Cohenour (Albuquerque); and the following members: C. F. Milligan (Clayton); C. A. Miller (Las Cruces); G. W. Jones (Clovis); W. T. Joyner (Roswell); H. A. Ingalls (Roswell); M. K. Wylder (Albuquerque); W. H. Woolston (Albuquerque); H. A. Miller (Clovis); R. O. Brown (Santa Fe).

Minutes of the meeting of May 19, 1932, were read and, no objections arising, were ordered approved as read.

The Committee on Necrology submitted its report, as follows: "Resolved: That the New Mexico Medical Society notes with regret the passing from this life of the following: Dr. Jesse G. Holmes, Alamogordo; Dr. Daniel H. Lewis, Albuquerque; Dr. E. D. McKinley, Alamogordo; Dr. J. O. Hatcher, Deming.

(Signed) M. K. WYLDER,
ROBERT O. BROWN,
F. H. CRAIL."

Dr. W. T. Joyner (Roswell) discussed the importance and necessity for the adoption of an amendment to the By-Laws whereby the Council would be empowered to look out for the matter of dealing with public health and legislation insofar as it might pertain to the best interests of the medical profession and people. He urged this because of the fact that, before another State meeting will be held, there will be a Legislative session, stating that in the past years the Society has done little toward legislative matters pertaining to public health. Action should be taken looking toward legislation, perhaps not so much in promoting legislation, as in preventing vicious legislation from being enacted. Since the osteopaths and chiropractors have become legalized in this State, they have become militant and are asking for more and more each year. Under our present system, the By-Laws provide for a Committee on Public Health and Legislation, which committee is appointed by the President on recommendation of the county societies. This has not been a practicable method by which to deal with the situation, as the committee is shifted around each year and it is difficult to get the members together for action which may be desired or necessary at any time. The Council, as the executive body of the Society in the interim between meetings, should have this duty devolve upon it, as it is usually composed of members who have belonged to the Society for years, who attend the meetings regularly (most of them being ex-Presidents), it seems best fitted to look after these important matters.

After considerable discussion, Dr. W. T. Joyner (Roswell), made motion that Section 3 of Chapter 8 of the By-Laws be amended. This Section now reads: "The Committee on Public Policy and Legislation shall consist of a member of each component Society, appointed by the President on the advice of and confirmed by the Council, and the President and Secretary-Treasurer. Under the direction of the House of Delegates, it shall represent the Society in securing and enforcing legislation in the interest of public health and of scientific medicine. It shall keep in touch with professional and public opinion, shall endeavor to shape legislation so as to secure the best results for the whole people, and shall strive to organize professional influence so as to promote the general good of the community in local, state, and national af-

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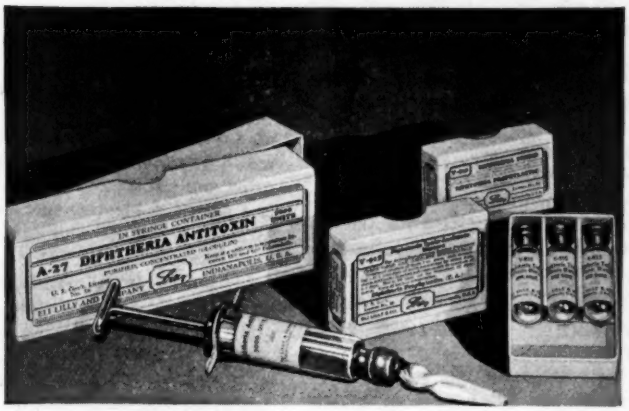

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fairs and elections." The proposed amendment would strike out the words "it shall represent the Society", and in their place substitute the words, "it shall assist the Council."

Further, to Chapter 7, which provides the duties of the Council, should be added a Section to be known as "Section 6", to read: "The Council shall represent the Society in securing and enforcing legislation in the interest of public health and scientific medicine. It shall keep in touch with professional and public opinion, shall endeavor to shape legislation so as to secure the best results for the whole people, and shall strive to organize professional influence so as to promote the general good of the community in local, state and national affairs, and elections."

Dr. Joyner stated that there would still be the Public Health Committee to assist the Council, and it would be the duty of the Council to take up all matters pertaining to public health and legislation which might arise at any time between meetings. He announced that, if the proposed amendments were adopted, he intended to offer the following Resolution: "Resolved, That the sum of Five Hundred Dollars ($500.00), is hereby appropriated for the use of the Council, to be expended as it may be deemed necessary during the ensuing twelve months in the interest of legislation pertaining to public health and scientific medicine, and pay the expenses of holding special meetings in the interests of the Society, and it is hereby requested that the Council hold at least one special meeting during the 1933 session of the New Mexico Legislature, to consider such matters and take such action as it may deem necessary."

The motion was seconded by Dr. R. O. Brown (Santa Fe) but, under the rules, had to be held over until next meeting.

Election of officers followed:

President-Elect: Dr. H. A. Ingalls (Roswell) was nominated by Dr. M. K. Wylder, seconded by Dr. W. T. Joyner. Motion that nominations be closed was seconded and unanimously carried. Dr. Ingalls was declared elected.

Vice-President: C. F. Milligan (Clayton) was nominated by Dr. W. T. Joyner, seconded by Dr. W. H. Woolston. Motion that nominations be closed was seconded and unanimously carried. Dr. Milligan was declared elected.

Secretary-Treasurer: L. B. Cohenour (Albuquerque) was nominated by Dr. H. A. Ingalls, seconded by Dr. M. K. Wylder. Motion that nominations be closed was seconded and unanimously carried. Dr. Cohenour was declared elected.

Councillors for Three Years to replace Dr. C. B. Elliott (Raton) and R. O. Brown (Santa Fe): Motion was made by Dr. L. B. Cohenour that Drs. Elliott and Brown be reelected to serve for three years more, seconded by Dr. H. A. Ingalls. Motion that nominations be closed was seconded and unanimously carried. Drs. C. B. Elliott and R. O. Brown were declared reelected.

Delegate to A. M. A. Meeting for 1933 and 1934: Dr. H. A. Miller (Clovis) was nominated by Dr. M. K. Wylder, seconded by Dr. H. A. Ingalls. After discussion as to the advisability of selecting different men for this office annually, or continuing the same incumbent because of his familiarity with procedure, motion that nominations be closed was seconded and carried. Dr. Miller was declared elected.

Alternate: Dr. J. W. Board (Clovis) was nominated by Dr. W. T. Joyner, seconded by Dr. H. A. Ingalls. Motion that nominations be closed was seconded and unanimously carried. Dr. Board was declared elected.

Meeting Place for 1933: Dr. W. T. Joyner extended a cordial invitation for the Society to hold its 1933 meeting at Roswell. The Secretary read telegrams from the Roswell Chamber of Commerce urging that the Society meet at that place in 1933, and also from the Carlsbad Chamber of Commerce, urging that Carlsbad be selected as the meeting place. Motion by Dr. M. K. Wylder that the 1933 meeting be held at Roswell, was seconded by Dr. Cohenour and carried.

Dr. H. A. Ingalls (Roswell) made motion that the Secretary be instructed to notify the Chamber of Commerce at Carlsbad, of the appreciation of the Society of their invitation, which it regretted could not be accepted this year because Carlsbad had been the meeting-place since Roswell and therefore it seemed Roswell should have the next meeting. Seconded by Dr. W. T. Joyner and carried.

Motion by Dr. W. T. Joyner that the meeting be adjourned until 8:30 a. m., May 21, was seconded and carried.

SATURDAY, MAY 21, 1932
House of Delegates

The meeting was called to order at 9 a. m., by the President, Dr. F. D. Vickers. There were present: Dr. F. D. Vickers (Deming); Dr. L. B. Cohenour (Albuquerque); Dr. C. F. Milligan (Clayton); Dr. C. A. Miller 'Las Cruces); Dr. H. A. Ingalls (Roswell); Dr. W. T. Joyner (Roswell); Dr. H. A. Miller (Clovis).

Minutes of the previous meeting were read by the Secretary and, no objection arising, were approved as read.

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Dr. W. T. Joyner (Roswell) requested that the Secretary read the proposed Amendments to the By-Laws, as brought before the meeting yesterday. After the reading, motion was made by Dr. H. A. Ingalls (Roswell), that the proposed Amendments as read by the Secretary, be adopted, which was seconded by Dr. H. A. Miller (Clovis) and carried.

Dr. W. T. Joyner (Roswell) offerel "Resolution, That the sum of Five Hundred Dollars ($500.00) be appropriated for the use of the Council, to be expended as it may be deemed necessary during the ensuing twelve months in the interest of legislation pertaining to public health and scientific medicine, and pay the expenses of holding special meetings in the interests of the Society, and it is hereby requested that the Council hold at least one special meeting during the 1933 session of the New Mexico Legislature, to consider such matters and take such action as it may deem necessary."

Motion was made by Dr. H. A. Miller (Clovis) that Resolution as offered by Dr. Joyner to appropriate $500 for use of the Council as stated, be adopted. Seconded by Dr. H. A. Ingalls, Roswell, and carried.

Dr. W. T. Joyner (Roswell) made motion that the Secretary be instructed to have printed as many copies as he might deem necessary of the Constitution and By-Laws, as amended, and that each member of the Society be furnished with a copy. Seconded by Dr. C. A. Miller (Las Cruces) and carried.

No further business arising, motion to adjourn was carried, with adjournment at 9:15 a. m.

Meeting of Council

Called to order by the President, Dr. F. D. Vickers, at 9:15 a. m., there were present in addition to Dr. Vickers, the following: Dr. L. B. Cohenour (Albuquerque); Dr. W. T. Joyner (Roswell); Dr. H. A. Miller (Clovis), and Dr. R. O. Brown (Santa Fe).

After discussion of the Amendments to the By-Laws, Dr. H. A. Miller made motion that the President and Secretary-Treasurer of the Society, act as President and Secretary of the Council and call such meetings as may be deemed necessary prior to the meeting of the New Mexico Legislature. Seconded by Dr. Joyner and carried.

Motion was then made by Dr. W. T. Joyner that the President of the Council call a special meeting at Albuquerque during the meeting of the Southwestern Medical and Surgical Association (probably sometime in November or early December), for the purpose of working out a program for the ensuing year along legislative or other lines. Seconded by Dr. R. O. Brown and carried.

Dr. W. T. Joyner (Roswell) made motion that the Secretary be instructed to write to each county society secretary in regard to ascertaining the views of prospective candidates for Legislature, and whether they would be favorable or otherwise to medical legislation, if elected. Seconded by Dr. H. A. Miller and carried.

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No further business arising, motion to adjourn was entertained and carried. Adjournment 9:30 a. m.

Scientific Session

Dr. R. A. Duncan (Amarillo, Texas) presented his credentials as Fraternal Delegate from the State of Texas, and issued a cordial invitation for members to attend the Texas State meeting next year.

Dr. James W. Hendrick (Amarillo, Texas) presented a paper on "Diagnosis and Management of Adenomatous Goiter", in which he pointed out that there are two types of adenomata: the fetal type, which is the less common of the two, and the so-called colloid adenomata. Clute states that 90 per cent of the thyroid cancers have their origin in this type of tumor, and that at least 4 per cent of the fetal adenomas become malignant. The colloid adenomatous goiter occurs more frequently in the so-called goiter belts, but sporadic cases are observed throughout the United States. This form of goiter, once present, is strictly a surgical condition, as it is not amenable to medical treatment. Its course is constant and progressive, and does not run in cycles like that of the exophthalmic. About 60 per cent of the adenomas become toxic in middle life. Influenza, tonsillitis, quinsy, bronchitis, physical strain, mental shcok, and pregnancy, may act as predisposing factors of toxicity in many cases. Whereas, in other cases, toxicity develops without any apparent stimulating factors.

Discussion was opened by Dr. W. S. Lemon (Rochester, Minn.), who advised surgical treatment in all cases of adenomatous goiter in which the adenomas are at least an inch in diameter. If there is a single adenoma, circumstances may be such as to make its removal advisable at any age; if multiple adenomas are present, it is usually advisable to wait until the patient is over twenty-five years of age. Multiple adenomas are, in all probability, better removed in all patients over forty years of age. They apparently do not begin to hyperfunction until after an average of fourteen years has passed following discovery.

Dr. F. D. Vickers (Deming) asked in regard to the value of x-ray treatment, especially in the case of small adenomas that are highly secretive.

In closing, Dr. Hendrick advised that the time for removal of non-toxic adenomas had moved up to around the age of thirty, as, if removed too early, very small adenomas might be overlooked, or new adenoma might develop and later give trouble. X-ray treatment he did not advise in any type of adenoma.

Dr. R. A. Duncan (Amarillo, Texas), in a paper on "Brain Abscess from the Otologist's Viewpoint", called attention to the status of brain abscesses as seen by the average physician, presenting cases taken from the records of Saint Anthony Hospital and Northwestern Texas Hospital, Amarillo, Texas, which were seen and diagnosed by many different types of practitioners, the records indicating two things: (1) the inclination to procrastination on the part of the patients and the hopelessness of the condition during the late stages; (2) the advantage that could be had from having trained neurologists and brain surgeons in all hospitals. In the cases recorded, Dr. Duncan stated, the diagnosis was made too late to be of any benefit to the patient. "They show the value of early diagnosis if surgery is to do anything other than hasten the end. Someone has very aptly said that, 'when a diagnosis is made of brain abscess, it is too late to operate.' The symptoms must be interpreted early if the patients are to be benefited. The classical symptoms cannot be waited for. When they appear, the case has progressed to a dangerous stage. It is important that we keep in our minds the potential danger of a chronic discharging ear, so that, when there is any diviation from its normal course, such as headache, vomiting, loss of smell, muscular weakness and other symptoms of this type, we shall be on our guard. Early diagnosis and prompt treatment are essential if we are to aid the patient."

In discussion, Dr. J. P. Kanes (Santa Fe) complimented Dr. Duncan on his intensive study and portrayal of the cases cited, referring to Sir James Mackenzie and the manner in which he studied the heart, as a comparison.

Dr. Duncan, in closing, thanked the Society for the opportunity of presenting his paper and in-

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timated that he, like Mackenzie, was a Scotchman, but could appreciate Scotch jokes nevertheless.

Dr. E. B. McBride issued a very cordial invitation to members to attend a fall clinic to be given at Oklahoma City, the first week in November, when an excellent program will be presented.

Motion pictures on spinal anesthesia followed the conclusion of the scientific program.

General Session

A meeting of the general session was held immediately following adjournment of the scientific session, President Vickers presiding. After call to order, the Secretary's report, including a resume of the proceedings of the House of Delegates (which has been published in full in the minutes of the meeting), was read and adopted.

Dr. C. F. Milligan (Clayton) proposed a Resolution that thanks be extended to the Governor of New Mexico, the Mayor of Santa Fe, the Santa Fe County Medical Society, the Program Committee, the Entertainment Committee, the Committee on Arrangements, Director and Mrs. Nusbaum of the Laboratory of Anthropology, and, in addition, the management of the La Fonda Hotel, the members of the press, the Ladies' Auxiliary, and the management of the Lensic Theatre, for the very cordial hospitalities extended and courtesies shown the members and visitors, which assured success for the meeting. The motion was seconded and carried.

Dr. W. T. Joyner (Roswell) made motion that the President appoint Fraternal Delegates to attend the annual meetings of the Colorado, Texas, and Arizona State Societies. Seconded by Dr. R. A. Miller (Clovis) and carried.

The President announced as such Fraternal Delegates:

To Texas State Meeting, Dr. H. A. Ingalls, Roswell.

To Colorado State Meeting, Dr. T. P. Martin, Taos.

To Arizona State Meeting, Dr. G. T. Colvard, Deming.

In closing the session, President Vickers thanked members for their presence and interest manifested in the proceedings.

Adjournment sine die at 12:15 p. m.

BOOK REVIEWS

COURTS AND DOCTORS, by Lloyd Paul Stryker; The MacMillan Co., New York 1932. $2.00.

This is a book which every physician should read. It is written by a lawyer who has had a great deal of experience in the trying of malpractice suits in the New York state. He for years represented the New York State Medical Society.

The book contains 236 pages and is divided into seven parts. The first defines the practice of medicine; the second deals with relationship of patient and physician, especially emphasizing confidential communications; part three, with the action for malpractice; four, the defenses to actions for malpractice; five, with expert testimony; six, the doctor

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on the witness stand; and, seven, the doctor and the criminal law.

In part three the author goes into the detail of what is apt to constitute malpractice and what is necessary to prove when establishing a suit for malpractice. He says that it is practically always essential, in order to have a successful suit for malpractice, to have expert testimony to the effect that the defendant did not use reasonable care and reasonable knowledge compared with others' practice in his community. There are exceptions to the expert testimony rule. It is possible for a patient to acquire an x-ray burn and be able to exhibit the burn to the jury and establish that it came from use of the x-ray. This might take the place of an expert in such cases.

The book is well written. The introduction is a splendid piece of literature and is well worth frequent perusal.

On page 15 of the introduction, 5th line from the bottom, the author says that Dr. Morton had been experimenting for many years with sulphuric acid, and he must mean sulphuric ether. On page 88, in the 5th line of the second paragraph, he says that "Dr. Myers removed the right kidney and the bowel"; evidently he has not said just what he means. On page 93, top line, he uses the word anesthesia when he really means anesthetic. Again, on page 112, last paragraph, and page 113, first paragraph, he uses anesthesia when he really means anesthetic. On page 155, 7th line from the top, he uses words "an expert" when he should have said experts.

On page 114 there is a paragraph which every physician should memorize. It is one of eleven suggestions to avoid malpractice suits. It reads as follows: "Be tactful and just to your fellow practitioners. Do not indulge in needless criticism. The fact that you form or act upon a conclusion different from that of your predecessor affords in and of itself no just basis for condemning his judgment or his action. Careless remarks, ofttimes unjust, have not infrequently led to litigation."

This is a day when everybody is looking for easy money or any kind of money and in which there is an epidemic of malpractice suits, attempts to filch doctors.

May every physician read this little book.

O. H. B.

COLLECTED PAPERS OF THE MAYO CLINIC AND THE MAYO FOUNDATION; edited by Mrs. Maud H. Mellish-Wilson and Richard M. Hewitt, B. A., M. A., M. D.; Volume XXIII 1931; Published, May 1932; Philadelphia and London; W. B. Saunders Company.

The 1931 edition contains 1231 pages. There are about 265 to 270 contributors and about 910 separate articles. The subject matter runs the gamut of scientific medicine and it is undoubtedly the outstanding yearly collection of contributions. One who has to keep up with the progress of medicine must have access to these volumes. There are so many interesting articles that it is very difficult to select any one to be commented upon.

The one which is outstanding in the mind of the reviewer is entitled "Studies of the Physiology of the Liver. XXII. The van den Bergh Reaction in the Jaundice Following Complete Removal of the Liver". The article gives the theories of the van den Bergh reaction and a conception of it that I had been unable to gather from any other source. Bilirubin probably undergoes a change as it passes through the polygonal cells of the liver, and this change is responsible for a quick reaction to the diazo reagent whereas before the reaction is slow. Thus we have the direct and the indirect reactions. It is an interesting thing that the kidney cells of the dog seem to have the same effect on bilirubin as do the liver cells of man. The broken down hemoglobin is transformed probably into bilirubin in the reticulo-endethelial system.

Some of the most interesting articles in the volume deal with the subject of medicine in a more or less general way. The writers of these are mainly Drs. W. J. Mayo, C. H. Mayo, Judd, Wilson and Walters.

One almost sheds a tear when reading Wm. J.'s statement "my brother, Chas. H. Mayo is not only the best clinical surgeon from the point of the pa-

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CRUM EPLER, M. D.
Superintendent

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tient that I have ever known, but he has that essential attribute of the true gentleman, consideration for others". He said this on the anniversary of his 70th birthday when speaking of the delightful association he had had with his brother these many years. He said that they had a common pocket book in which each has wanted the other to have the greater share.

O. H. B.

SURGICAL PATHOLOGY OF THE FEMALE GENERATIVE ORGANS, By Arthur E. Hertzler, M. D.; Surgeon to the Agnes Hertzler Memorial Hospital, Halstead, Kansas; Professor of Surgery, University of Kansas; 285 Illustrations; J. B. Lippincott Company; Philadelphia, Montreal and London.

Those who are acquainted with Dr. Hertzler need no words of mine to give them a concept of what this volume contains. It is a result of his large experience. In his preface he says, "I have relied on that master critic in anatomic diagnosis, the after-course, to check the histologic diagnosis".

It is such works as this, the experiences of well-trained individuals, which will advance and further perfect the various divisions of the science of medicine. Every man doing general surgery and every gynecologist should own this book.

There are three parts to the book, to-wit: Diseases of the external genitalia in the female; Diseases of the tubes and ovaries; Diseases of the uterus. There are two chapters in the first part, four chapters in the second and nine in the third.

He seems to have practically covered every possible pathological development of the female generative organs.

Hertzler has a direct way of saying things so that his meaning is clear and usually is easy to understand. The sentences are ordinarily short. His language is impressive. For instance on page 71 he says, "It has always been a mystery to me why surgeons should regard the castration of the human female with indifference, even derision. Even now women come to the clinic in droves who have had their ovaries removed and were wrecked by it." On the next page he says, "Formerly it was the surgeons who castrated their patients, now it is the roentgenologists. I will have no more of it. There is not a single lesion described in this chapter which can be maintained as pathologic in a degree that justifies removal and none in which the removal of the ovary but makes the last state of the patient worse than the first".

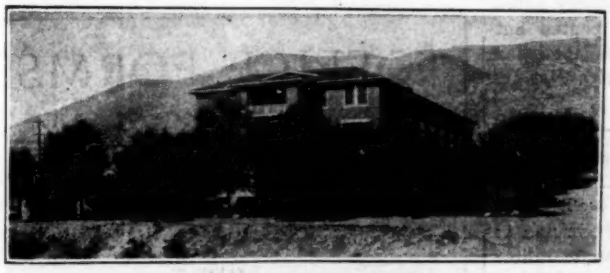
In places his language becomes a bit satirical but it is Hertzlerian and could not be otherwise. The publishers have done their work almost, if not quite, to perfection. The index appears to be well arranged. Let these reports, for such they are, of Hertzler's, be a stimulus to others who have had extensive experience coupled with original thinking.

O. H. B.

MEDICAL CLINICS OF NORTH AMERICA: (Issued serially, one number every other month); Volume 16, Number 1; (Philadelphia Number—July 1932); Octavo of 290 pages with 75 illustrations; Per Clinic year, July 1932 to May 1933, Paper, $12.00; Cloth, $16.00 net.; Philadelphia and London; W. B. Saunders Company, 1932.

There are a number of articles in this issue which practically every physician would wish to read in their entirety; Psychological Considerations in Schizophrenia, by Edward A. Strecker; Can We Prevent Heart Disease, by James E. Talley; Injury to the Spinal Cord, by eGorge Wilson; Chroniv Infection, by Martin E. Rehfuss; Prevention of Coronary Occlusion, by Wm. D. Stroud; Alkalosis and Duodenal Ulcer, by H. L. Bockus and J. Bank; Purpura Hemorrhagics, by Harold W. Jones and Leandro M. Tocantins are among the more interesting ones.

Rehfuss in his article on chronic infection has been studying bacteria, isolated in patients, for their grade of pathogenicity. In order to determine whether a certain streptococcus is producing pathologic changes he determines the evidence of local reaction for the presence of organisms where they should not be, the skin reactions to the vaccines, the complement fixation test, and the pathogen selectivity test.



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Medical Director

He is inclined to the opinion that the intradermal tests are of definite value in deciding upon the likelihood of a streptococcus being to blame for trouble. He points out that two organisms may be morphologically identical and one may be nonpathogenic while the other is highly virulent.

The article is worth the price of the volume. Other articles in this issue are just as valuable.

O. H. B.

NEW AND NONOFFICIAL REMEDIES, 1932, containing descriptions of the articles which stand accepted by the Council on Pharmacy and Chemistry of the American Medical Association on Jan. 1, 1932. Cloth. Price, postpaid, $1.50, Jp. 402. lvi. Chicago: American Medical Association.

The recognition of a preparation for inclusion in this book singles it out from the host of new products of the pharmaceutical manufacturers as being a worthwhile addition to the existing armanentarium of the practicing physician. To be thus distinguished it must be shown, under the impartial scrutiny of the carefully chosen group which is the Council on Pharmacy and Chemistry, that it has acceptable evidence of therapeutic usefulness and that it is marketed in accordance with the honesty and straightforwardness envisaged by the excellent Rules which have been the outgrowth of the Council's quarter century experience in appraising the merits of new drugs.

In accordance with its custom of keeping the annual editions of New and Nonofficial Remedies in the forefront of current medical thought, the Council offers in this volume the newly revised articles: Barbital and Barbital Compounds; Fibrin Ferments and Thromboplastic Substances; Liver and Stomach Preparations; Mercury and Mercury Compounds; and Ovary. Perhaps the most noteworthy new preparations admitted are: nupercaine-Ciba, a local anesthetic; pentobarbital sodium, a barbituric acid derivative; and iopax, a new preparation for roentgenologic use. All of the ovary preparations formerly described are omitted and none of the new standardized preparations are described, although the names Theelin and Theelol are recognized in the revised general article. Another change of importance is the classification of articles formerly listed as "Exempted" under the heading "Accepted but Not Described." There is the usual excellent index and the augmented Index to Proprietaries Not Included in N. N. R.

A TEXT-BOOK OF MEDICINE; by American Authors. Edited by Russell L. Cecil, A. B., M. D., Sc. D.; Assistant Professor of Clinical Medicine in Cornell University; Assistant Visiting Physician to Bellevue Hospital, New York City; Associate Editor for Diseases of the Nervous System; and Foster Kennedy, M. D., F. R. S. E.; Professor of Neurology in Cornell University; Head of Neurological Department, Bellevue Hospital, New York City. Second Edition, revised and entirely reset. W. B. Saunders Company, Philadelphia and London, 1930.

The reviewer gets the impression that this book should take the place once held by that splendid book on the Practice of Medicine by the great American clinician—Osler. When one pages through it and reads paragraphs here and there, it seems that just the right things and the right amounts are said to give students the view they should have of the disease in question. It does seem that a more careful editing of the text could have eliminated many words and probably shortened it considerably; for example, on page 39 in the third paragraph, in the last line, the word "any" and "all" might well have been eliminated without weakening the statement. In the last paragraph the words "at all" might well have been eliminated. In the last paragraph page 366, the word "some" is used twice without increasing the strength of the statement, and again, the words "any" and "very" are used when they might well have been eliminated. Such superfluous words are found throughout the text. They do not detract from the value of the book; they do, however, mar its beauty of expression.

O. H. B.

A DIAGNOSTIC PROBLEM

(Taken from the New York Sun.)

(Problem: What is the name of the engineer mentioned below? The answer can be ascertained by careful analysis of the statements made.)

A railroad train had a crew of three, and three passengers, traveling between Chicago and New York. The train crew is made up of an engineer, a fireman and a guard. Their names are Smith, Jones and Robinson, but not necessarily in that order. The passengers have the same names, but will be called Mr. Smith, Mr. Jones and Mr. Robinson.

Mr. Robinson lives in New York. Mr. Jones' annual salary is $5,000. The guard lives half way between New York and Chicago, and his namesake among the passengers lives in Chicago. The guard's closest neighbor is one of the passengers and his annual salary is exactly three times that of the guard. Smith beat the fireman at billiards.

Diagnostic analyses are invited.

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